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STUDIES ON LILY VIRUS DISEASES: THE NECROTIC-FLECK COMPLEX IN LILIUM LONGIFLORUM

PHILIP BRIERLEY[1] AND FLOYD F. SMITH[2]

(Accepted for publication January 10, 1944)

The necrotic fleck disease in Easter lilies (*Lilium longiflorum* Thunb.) is capably described in an early account by Stewart (19) who distinguished it from Botrytis blight, and found the necrotic streaks were not due to insect or fungus attack. Necrotic fleck symptoms are included in Woods' account (22) of "The Bermuda Lily Disease," and illustrated in a second paper by that author (23). Neither of these early workers suggested the virus etiology of the disease.

Ogilvie and Guterman (17) described a mosaic "disease or group of diseases" in Bermuda Easter lilies, distinct from the rosette disease (or yellow flat) (5, 16). They distinguished 3 types of symptoms: A. Marked downward curling of leaves accompanied by short chlorotic streaks becoming rusty necrotic, and flower distortion; B. Similar linear necrotic spotting, with some twisting but no characteristic curling of leaves, and no marked distortion of flowers; C. The leaves mottled, notably stiff and variously distorted, and the flowers aborted or deformed. Ogilvie and Guterman (17) recognized these diseases as of virus origin, and observed evidence of natural spread in Bermuda, but were unsuccessful in attempts to transmit them. Symptom types A and B of these investigators correspond well with the severe and the mild forms, respectively, now recognized in necrotic fleck, but their type C, possibly due to a mottle virus (4), cannot be definitely classed from their description and illustrations.

Guterman first transmitted "the virus of lily mosaic" by inarching and by needle-prick (8), and by *Aphis gossypii* Glover (9, 10). He recognized 3 types of mottling in lilies: (1) linear, light-green areas, drying to more or less linear necrotic spots; (2) elongate chlorotic streaking without necrosis; and (3) chlorotic mottling in rounded or irregular areas without necrosis. The disease we now call necrotic fleck was shown as an example of the first type, and was considered the typical response of Easter lily, while the characteristic symptoms of over 40 other species were assigned to one or another of the above 3 types. Extensive cross-inoculations between species by stem puncture, and a number of successful transfers by *A. gossypii*, seemed to yield uniform symptoms of some one of these 3 types in each species inoculated, regardless of the species serving as source of virus. Guterman (9), therefore, concluded that "No evidence of any sort was obtained during these experiments which would indicate that more than one

[1] Pathologist, Division of Fruit and Vegetable Crops and Diseases, Bureau of Plant Industry, Soils, and Agricultural Engineering.

[2] Entomologist, Division of Truck Crop and Garden Insect Investigations, Bureau of Entomology and Plant Quarantine, Agricultural Research Administration, United States Department of Agriculture, Beltsville, Maryland.

virus is concerned." He did observe, however, that "mosaic affected plants of *Lilium longiflorum* may not necessarily exhibit necrosis of the chlorotic areas on the leaves." Inoculations from various species showing symptom types 2 and 3, as well as from Easter lilies showing type 1, induced type 1 symptoms (*i.e.*, necrotic fleck) in certain of the Easter lilies inoculated. The fact that Guterman used symptomless commercial stock, rather than seedlings, as test material apparently prevented his recognizing the separate significance of mottling and necrotic flecking in Easter lily.

In 1937 Price (18) reported the isolation from commercial Giganteum Easter lilies of a cucumber-mosaic virus, which, in common with other strains of this virus, was capable of inducing necrotic fleck symptoms in certain Easter lilies that "were either grown from seed or were from selected cage-grown stock." Although the necrotic symptom appeared in a low percentage of the Easter lilies he inoculated with cucumber-mosaic virus, Price accepted Guterman's (10) interpretation of lily mosaic as being of single virus etiology and assigned this lily-mosaic virus to the cucumber-mosaic virus group.

McWhorter was the first to show tulips are susceptible to viruses from lilies, reporting tulip breaking induced by juice of mosaic *Lilium speciosum* in 1932 (12), and in 1937 (13), by juice of symptomless *L. candidum* L., *L. longiflorum*, and *L. tigrinum* Ker-Gawl. His latent virus of lily at first (13) considered identical with Tulip Virus 1 (14) was later (15) distinguished from this by cytological methods.

One of us (1), impressed with the distinctness of mottle and necrotic fleck symptoms in the Creole Easter lily, repeated Price's (18) and McWhorter's (13) experiments, finding a tulip-breaking virus in both mottled and flecked Easter lilies, but cucumber-mosaic virus in the necrotic fleck type only. Subsequent work (3) showed that cucumber-mosaic virus alone does not induce necrotic fleck in previously virus-free Easter lily seedlings, but does produce this symptom when introduced into certain symptomless commercial Easter lily stocks. Tulip breaking induced in tulips by cucumber-mosaic virus was also shown to be recognizably different from the type produced by the true tulip-breaking viruses and their lily-mottle allies. Brierley (2) then showed that seedlings of *Lilium formosanum* Stapf are satisfactory index plants for the tulip viruses and, using these and tobacco as test subjects, found both cucumber-mosaic virus and tulip-type viruses widely distributed in lilies, the latter being more common than the former.

Further studies on the lily-mottle viruses or tulip-breaking types will be presented separately (4). The present paper presents evidence that the necrotic fleck disease (NF) is a complex of cucumber-mosaic virus (CV) and a symptomless virus (LSV) not previously reported, that *Aphis gossypii* is a specific vector for LSV, and therefore for the complex CV + LSV, and that mottle viruses (LMV) consistently associated with necrotic fleck in commercial Easter lilies are not an essential part of the fleck complex. Some information is included on the distribution, host range, and economic impor-

tance of LSV, termed lily-symptomless virus and described as *Adelonosus lilii*, nov. gen. et sp.

Preliminary evidence is presented that LSV and lily-rosette virus (5, 16) are of the "persistent" group (20), having latent periods of several days in their common vector *Aphis gossypii;* but that lily-symptomless virus (LSV) does not protect Easter lilies against infection by lily-rosette virus (LRV).

SYMPTOMS OF NECROTIC FLECK

As previously mentioned, Stewart (19) and Guterman (9, 10) have described this disease. The term "fleck" was applied by one of us (1) in order to provide a distinctive name for the complex. Characteristic flecks are variable in size, usually elongated parallel to the veins, chlorotic when first recognized but becoming gray to brown necrotic (Fig. 1). When fully developed the dead areas are depressed but the surface remains intact. The name necrotic fleck (NF) is reserved for this complex in *Lilium longiflorum* in our usage. Other lily species may develop necrotic spotting, as noted by Guterman (9, 10), but only the Easter lily has thus far been shown to develop these symptoms from infection by the virus complex here discussed.

Dwarfing, curling (Fig. 2), and flower deformities (Fig. 3) also accompany the typical NF. The stature is often less than half that of symptomless plants of the same variety. Leaves may be curled downward as well as flecked, but this curling is less regular and commonly less extreme than that characterizing lily rosette disease (16). Flowers are subnormal in size, fail to open fully, and are variously distorted by thin streaks which become brown necrotic by the time the flower is fully developed (Fig. 3). Such typical NF individuals, comparable to Type A of Ogilvie and Guterman (17), are unsalable. After flowering, and sometimes before, NF plants begin to lose leaves from below upward. Such leaves first turn pale-yellow with irregular green blotches, then wither away. Affected plants mature earlier than symptomless individuals of the same variety, and yield small bulbs. Such bulbs are subject to rot, and the survival of the NF type is low. The NF symptoms always persist, in those individuals that do survive, during the next season of growth.

A milder sub-type, scored as "sparse fleck" (SpF) in our records, is comparable to type B of Ogilvie and Guterman (17). In this type the necrotic flecks are fewer and usually larger than in the severe type, there is less marked dwarfing, the leaves are twisted irregularly instead of curled downward, and the flowers are seldom distorted. SpF plants are often considered salable, particularly in years of deficient supply. Experimentally produced flecking is often of this SpF type (Fig. 4), and the illustrations Price (18) published may be so classed. SpF Easter lilies grown in screened and fumigated greenhouses for several successive seasons are fairly stable in symptom expression, but some NF may develop in these under such conditions.

A still milder type, which we term "Very Sparse Fleck" (VSpF), may be detected in certain commercial Easter lilies under glass, but is doubtless

overlooked in the field where various injuries may be confusing. No dwarfing, curling, or flower distortion accompanies the very sparsely distributed necrotic flecks; affected plants are, therefore, salable. The VSpF type selected from commercial Creole lilies forced in the season of 1939–40 and

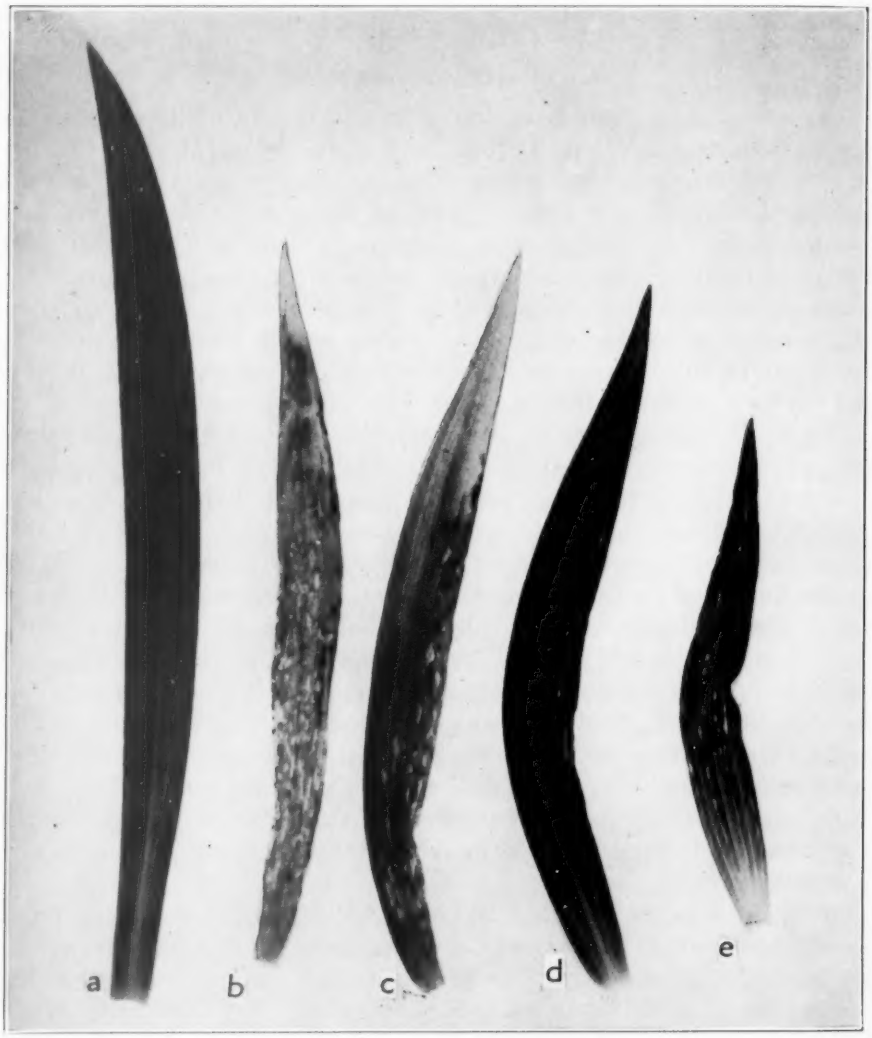

FIG. 1. Leaves of an Easter lily seedling experimentally infected with necrotic fleck by *Aphis gossypii:* (a) normal lower leaf, (b) first of the group of leaves showing advanced necrosis and yellowing, (c, d, e) younger leaves in less advanced stages of fleck symptoms. Photographed by Pratt.

grown again under glass in 1940–41 produced 1 symptomless individual, 1 VSpF, and 15 severe SpF plants, of which 13 were judged unsalable because of flower distortion. Ten VSpF plants selected from forced Creole lilies in 1941–42 and grown again under glass in 1942–43 produced 3 symptomless, 3 VSpF, and 4 SpF plants, of which 3 were considered unsalable.

Attention is called to the two sub-types of necrotic fleck, SpF and VSpF, as confusing factors in the estimation of fleck in forced Easter lilies, and as obstacles to effective roguing in the field. Although examples of the 3 types of fleck described here can be found in nearly any affected commercial stock

FIG. 2. Necrotic fleck in Easter lily: (a, b) experimentally infected with cucumber-mosaic virus and lily-mottle virus in February, 1940, no fleck resulting. Lily-symptomless virus added to (a) by *Aphis gossypii* in February, 1941. Photographed in April, 1942; in 6-inch pots.

of Easter lilies, the sub-types are not separable one from another by sharp dividing lines, and all transition stages from the mildest VSpF to the most severe NF types occur. These types cannot be distinguished at present by their virus content, all 3 yielding CV and LMV on indexing, and all

evidently carrying LSV which is now held essential to the expression of NF symptoms. Attempts to demonstrate another factor for dwarfing in the severe NF type have not been successful, and the virulence of the complex has not been found consistently correlated with the virulence of the CV component.

The milder necrotic fleck types (SpF, VSpF) are commonly ignored by producers of Creole Easter lilies, who recognize the severe or typical NF type

FIG. 3. Symptoms of necrotic fleck in Creole Easter lily flower. The flower is small (4.5 inches long and 4.5 inches across), the perianth distorted and streaked with brown. Natural infection. Photographed by Allard.

only. It is true that this type only is consistent in producing cull types when forced, but the evidence presented above indicates that all 3 types represent essentially the same disease complex, and that shifts from the milder to the more severe types may be expected without additional contamination from outside sources.

MECHANICAL TRANSMISSION

Sap inoculation experiments of 5 categories are summarized in table 1. When cucumber-mosaic virus (CV) was inoculated into supposedly healthy Easter lily seedlings, only 1 plant of 512 developed necrotic fleck symptoms.

This one positive result may be discounted as contamination, inasmuch as the California-grown seedlings inoculated here showed 3 NF individuals in 750 plants previous to this test. It is reasonable to suppose that a few addi-

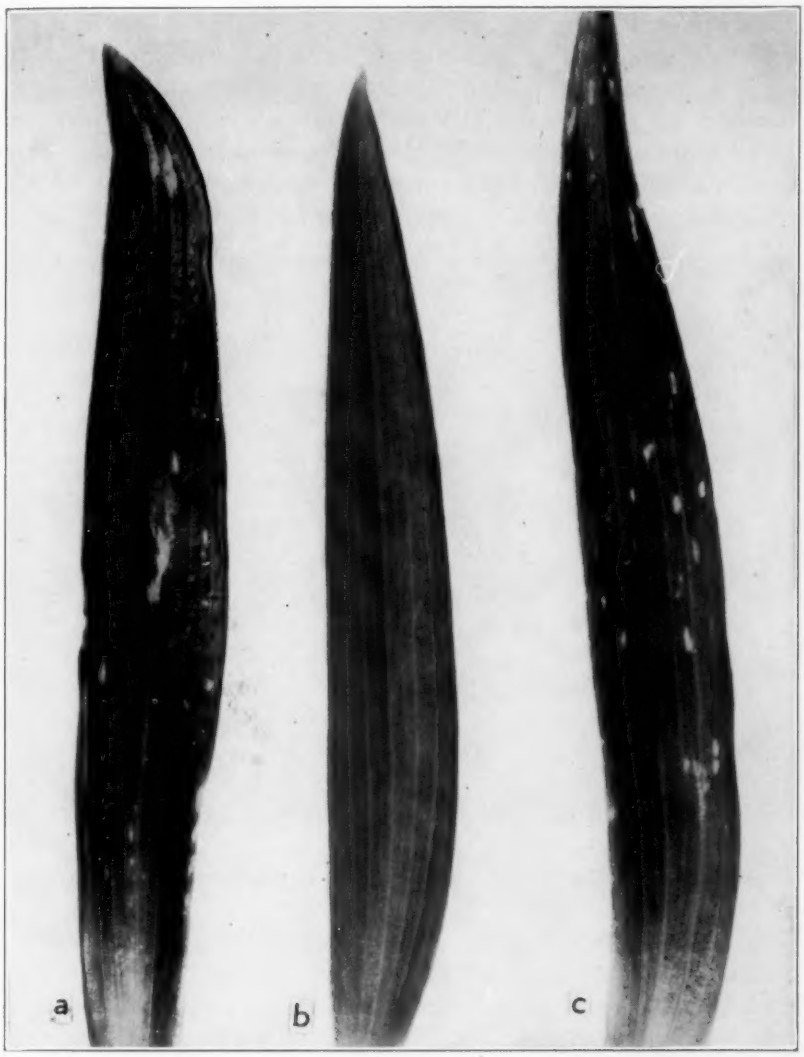

FIG. 4. Sparse fleck sub-type of necrotic fleck experimentally produced in Easter lily seedlings: (a, b, c) mechanically inoculated with cucumber-mosaic virus and lily-mottle virus in December, 1940. Lily-symptomless virus added by *Aphis gossypii* to (a) in March, 1941, and to (c) in January, 1942. Photographed in May, 1942 (natural size).

tional seedlings of this lot were contaminated with LSV which with CV produces NF. It is also reasonable to conclude that Price (18) was able to produce NF symptoms on inoculation with CV alone because some of the symptomless Easter lilies he inoculated carried LSV. The available evidence,

therefore, justifies the conclusion that CV alone cannot produce NF in healthy Easter lilies.

When CV was inoculated into symptomless commercial Easter lilies (LMV + LSV) a high proportion of necrotic fleck (97 in 132) resulted. Characteristic symptoms were thus produced in symptomless Creole Easter lilies (the Norwood strain and a Florida strain), and in symptomless Croft lilies, as well as in plants of Creole and Giganteum varieties that showed mottling. Inasmuch as all these commercial stocks, whether mottled or not, can be shown by indexing to carry LMV, the evidence thus far presented would justify the hypothesis that LMV and CV in combination produce NF. In some trials some strains of CV failed to induce NF under conditions appar-

FIG. 5. Necrotic fleck mechanically transmitted to Easter lily seedling (left) compared with control seedling (right). Photographed in 6-inch pots 55 days after inoculation.

ently favorable for infection, and cucumber appeared to be a poor source of CV for inoculation into lilies, but 100 per cent infection has been approached or attained in many such inoculations with more virulent strains of CV from tobacco.

Several combination inoculations with CV and LMV into healthy Easter lily seedlings failed to induce necrotic fleck. In separate trials preparations containing CV and LMV were mixed and inoculated simultaneously, or the two were introduced separately into different shoots of double-nosed Easter lily seedlings, or the one was introduced alone and then followed after 20 days by the other. No fleck resulted from any of these tests, although the inoculated seedlings were held for a year or more and subinoculations revealed both CV and LMV present. It was, therefore, concluded that the com-

bination of CV and LMV does not produce NF. It was further postulated that a third constituent (LSV), absent from suitably protected Easter lily seedlings but present in commercial Easter lilies, must be essential for the expression of necrotic fleck symptoms.

TABLE 1.—*Mechanical inoculation of Easter lily with necrotic fleck (NF), or with the cucumber-mosaic component (CV), or with CV and lily-mottle virus (LMV) together*

Source plant	Virus or complex	Plants inoculated	Total tests	No. of tests positive	Plants flecked[a]	Minimum incubation period
A. CV on healthy Easter lily seedlings						
						Days
Tobacco	CV	Easter lily sdlgs.	78	1	1/460	38
Easter lily	"	"	3	0	0/22	.....
Cucumber	"	"	2	0	0/30	.....
B. CV on commercial Easter lilies (LMV + LSV)						
Tobacco	CV	Creole (symptomless)	22	19	79/98	13
Easter lily	"	" "	1	1	5/5	20
Easter lily	"	" (mottled)	1	1	2/5	25
Easter lily	"	Croft (symptomless)	1	1	2/5	20
Cucumber	"	" "	1	0	0/5	.....
Tobacco	"	" "	2	1	4/9	33
Tobacco	"	Giganteum (mild mottle)	1	1	5/5	33
C. CV + LMV on healthy Easter lily seedlings						
Easter lily	CV + LMV	Easter lily sdlgs.	15	0	0/175	.....
Cucumber + E. lily	"	"	1	0	0/3	.....
D. NF on healthy Easter lily seedlings						
Commercial Easter lilies	NF	Easter lily sdlgs.	17	3	6/144	31
Easter lily sdlgs.	"	"	7	4	5/160	44
E. NF on commercial Easter lilies (LMV + LSV)						
E. lily sdlgs.	NF	Creole (symptomless)	3	3	23/25	13
"	"	" (mottled)	1	1	9/10	47
"	"	Croft (symptomless)	1	1	7/10	29

[a] Expressed as number flecked over number exposed (mottling not recorded here).

Necrotic fleck was sap-transmitted to healthy Easter lily seedlings in low proportion, 6 in 144 from naturally-infected source plants, and in about the same proportion, 5 in 160, from flecked material experimentally produced by previous mechanical transfer or by aphid transfer (Fig. 5). No infection resulted in trials in which the inoculum was prepared with phosphate buffers at pH 6, 7, or 8, or in saline solution, the successful transfers being pre-

pared with tap water. The low percentage of transfer (3.6 per cent) here recorded is considered authentic. Contamination of the test seedlings with the symptomless component (LSV) of fleck, as discussed above (Sect. A, Table 1), cannot explain the 11 positive flecks recorded here, for seedlings used in most of the positive tests were rigidly protected, some having been grown in screened and fumigated greenhouses throughout. Occasional seed-carriage of LSV may also be dismissed as an explanation of occasional transfer, as such seed-borne virus might be expected to occur with as great frequency in the 512 seedlings in Sect. A, of Table 1, or in the 178 in Sect. C, as in the 304 seedlings of Sect. D. It is therefore concluded that the NF complex is mechanically transmissible as such, but with difficulty. This difficulty of transfer is in itself further evidence for the existence of an additional essential component (LSV), as both CV and LMV are readily transferred mechanically.

Necrotic fleck is readily transferred to commercial Easter lilies of the Creole and Croft varieties, and presumably to other varieties not tested. This finding was to be expected as only CV is necessary to induce fleck in such stocks, as is shown above (Sect. B, Table 1). Comparison of the data summarized in Sections D and E of table 1 reveals a sharp contrast between the difficulty of transmitting NF to seedlings and the ease of transmitting it to commercial stocks carrying LMV + LSV. This contrast is held to be due to the nearly universal presence of LSV in commercial Easter lily stocks. Guterman's (9) ready transfer of a disease now interpreted as NF from Easter lily to Easter lily was evidently comparable to our experience (Sect. E, Table 1), although he used the Harrisii and what was probably Erabu (*Lilium longiflorum formosum*) varieties, which were not included in our tests.

TRANSMISSION BY APHIDS

Studies on vector relations of necrotic fleck and other virus diseases were undertaken in 1939. Some of the data on transmission of NF and its components CV and LSV are summarized in table 2. Transmission of the NF complex was determined by symptom expression in Easter lily, and transmission to other species was determined by return transfer to Easter lily by *Aphis gossypii*. Transmission of LMV was detected by characteristic symptom expression in Easter lily, *Lilium formosanum*, or tulip, or by mechanical subinoculation to one of the two plants last named. Transmission of CV was proved by subinoculation to tobacco.

In these experiments *Aphis gossypii* was confirmed as a vector of the NF complex (Fig. 6). Six other species of aphids failed to transmit this complex as such, but *Macrosiphum solanifolii* Ashm. and *Myzus persicae* Sulz. were found to transmit both CV and LMV. These findings are in agreement with those of sap transfer tests (table 1) in showing that CV and LMV together fail to produce NF, and that a further constituent (LSV) must be essential to expression of NF symptoms. They further indicate that *A. gossypii* is a specific vector of the LSV constituent, for this species is certainly not specific for CV.

Possible transmission of the NF complex to tulip, *Colchicum*, and *Gloriosa* is indicated in table 2. No distinctive symptoms were detected in tulip be-

TABLE 2.—*Transmission of necrotic fleck (NF), or of cucumber-mosaic virus (CV) or mottle virus (LMV) from flecked Easter lily to various plants by aphids*

Plants exposed	Tests		Plants affected[a]	Minimum NF incubation period	Virus transmitted		
	Total	Positive for NF			NF complex	CV	LMV
By *Aphis fabae* Scop.							
	No.	*No.*		*Days*			
Easter lily sdlgs.	1	0	0/6	.....	.....	.....	.....
Tulip (Clara Butt)	1	0	0/4	.....	.....	.....	.....
By *Aphis gossypii* Glover							
Easter lily sdlgs.	33	22	112/250	14	+	+	+
L. formosanum Stapf	3	0	0/16	.....	.....	+	+
L. tigrinum Ker-Gawl.	2	0	0/14	.....	.....	+	.....
Calochortus spp.	2	0	0/10	.....	.....	+	+
Colchicum autumnale L.	3	1	2/16	53	.....(?)	+	.....
Gloriosa rothschildiana O'Brien	4	3	14/25	10	.....(?)	+	.....
Fritillaria pudica Spreng.	1	0	0/5	.....	.....	+	.....
Crimson clover	1	0	0/5	.....	.....	+	.....
Tulip (Clara Butt)	7	5	28/78	21	.....(?)	+	+
By *Macrosiphum lilii* Monell							
Easter lily sdlgs.	7	0	0/45	.....	.....	.....	.....
L. formosanum	1	0	0/5	.....	.....	.....	.....
Tulip (Clara Butt)	1	0	0/8	.....	.....	.....	.....
By *Macrosiphum solanifolii* Ashm.							
Easter lily sdlgs.	8	0	0/85	.....	.....	+	+
Tulip (Clara Butt)	2	0	0/14	.....	.....	.....	.....
By *Myzus circumflexus* Buckt.							
Easter lily sdlgs.	3	0	0/26	.....	.....	.....	.....
L. formosanum	1	0	0/5	.....	.....	.....	.....
Tulip (Clara Butt)	1	0	0/17	.....	.....	.....	.....
By *Myzus convolvuli* Kalt.							
Easter lily sdlgs.	2	0	0/8	.....	.....	.....	.....
By *Myzus persicae* Sulz.							
Easter lily sdlgs.	12	0	0/116	.....	.....	+	+
Tulip (Clara Butt)	8	0	0/95	.....	.....	+	+

[a] Number of plants expressing NF over number exposed.

yond those known to be characteristic of CV infection, but two return transfers from the exposed tulips to Easter lily seedlings by *Aphis gossypii* pro-

duced typical NF. In *Colchicum autumnale* L. necrotic streaks appeared at the base of leaves in the region that was in active growth immediately after exposure (Fig. 7). CV was recovered mechanically, but a return transfer of *A. gossypii* to Easter lily seedlings yielded no NF. Attempts to induce these symptoms in *Colchicum* with CV alone have thus far proved unsuccess-

FIG. 6. Symptoms induced in Easter lily seedlings by aphid transfer from necrotic-fleck-infected Easter lilies: necrotic fleck transmitted by *Aphis gossypii* (a); lily-mottle virus transmitted by *Myzus persicae* (b); by *Macrosiphum solanifolii* (c), and by *Aphis gossypii* (d). Photographed by Pratt.

ful. *Gloriosa rothschildiana* developed striking yellow ring patterns in leaves after transfer of this vector from NF Easter lilies (Fig. 8). CV was recovered from such plants, and somewhat similar but less prominent rings were induced in *Gloriosa* seedlings by inoculation with CV only. One of 5 return transfers to Easter lily seedlings by *A. gossypii* produced NF. Unfortunately the apparently successful return transfers from both *Gloriosa*

and tulip were made to Easter lily seedlings that had been inadequately isolated in Southern California and carried some natural infection with NF and presumably also some additional infection with LSV. It is therefore uncertain whether necrotic fleck was returned to Easter lily from tulip and *Gloriosa* or whether CV was returned to seedlings already contaminated with LSV.

Cucumber-mosaic virus was recovered also from symptomless plants of *Lilium tigrinum, Calochortus* sp., and *Fritillaria pudica* exposed to fleck

FIG. 7. Symptoms induced in *Colchicum autumnale* by transfer of *Aphis gossypii* from necrotic-flecked Easter lily. Cucumber-mosaic virus was recovered. Photographed (in 6-inch pot) by Guernsey.

by *Aphis gossypii.* This virus was previously reported (2) as occurring naturally in *L. tigrinum. Calochortus* sp., *Colchicum autumnale, Gloriosa rothschildiana,* and *F. pudica,* are believed to be previously unreported as hosts of CV. The other plants listed as affected in table 2 have been previously reported susceptible.

The plants reported susceptible to lily-mottle virus in table 2 were previously known to be subject to this virus (4). In addition, *Lilium tigrinum* and *Fritillaria pudica* are susceptible; failure to develop symptoms in the tests recorded here is further evidence that *Aphis gossypii* is a poor vector of LMV. *Colchicum, Gloriosa,* and crimson clover have not proved to be susceptible to LMV.

In experiments conducted over a period of 4 seasons, *Aphis gossypii* transmitted NF, or its component LSV, to Easter lily seedlings somewhat irregularly. The irregularities cannot be fully explained, but a number of factors seems to be operative. First, all trials during the period of higher greenhouse temperatures from late April to early winter failed, even when succulent lateral shoots were exposed to the aphids. Secondly, *A. gossypii* seems unable to establish itself and thrive on the young leaves of Easter

FIG. 8. Symptoms induced in *Gloriosa rothschildiana* by transfer of *Aphis gossypii* from necrotic-flecked Easter lily (left) in comparison with control (right). Cucumber-mosaic virus was recovered. Photographed by Skeris.

lilies under the cooler conditions of fall and early winter, but more readily accepts leaves and flower buds in later stages of growth. Furthermore, even in winter and early spring, when transmission has been most consistent, the percentage of transfer has varied, suggesting that the concentration of one or both of the constituent viruses, CV and LSV, may vary in the Easter lily source plants.

Recent experiments show that LSV is of the "persistent" group as defined by Watson and Roberts (20), having a latent period of several days

in the vector *Aphis gossypii*, and persisting for at least 3 days in the vector on non-susceptible plants. Cucumber-mosaic virus, on the other hand, has been shown by Watson and Roberts (20) to be a "non-persistent" virus, surviving for a brief time in aphid vectors, and lacking a latent period in these. Inasmuch as the requirements for efficient transmission of viruses of these two groups are quite different, the transmission of a complex consisting of one of each group (CV + LSV) might be expected to be somewhat erratic. It was shown in one test that *A. gossypii* may transmit LSV

FIG. 9. Lily-rosette virus superimposed on Norwood Creole Easter lily naturally infected with lily-symptomless virus. *Aphis gossypii* transfer. Photographed (in 5-inch pot) 36 days after exposure.

without CV from NF source plants that carry both. One of 5 plants exposed developed NF symptoms, but the subsequent addition of CV to the remaining 4 plants induced one more to express necrotic fleck.

Lily-rosette virus (5, 16), received for diagnosis in Easter lily from Florida, was compared with LSV in some experiments. The data in table 3 indicate a latent period of 3 to 7 days in the aphid vector for lily-rosette virus (LRV), and one of 4 to 6 days for lily-symptomless virus. Similarly the data in table 4 show persistence of LSV in the vector *Aphis gosspyii* for at least 3 days, and of LRV for at least 9 days. The two viruses LRV and LSV are similar in most known characteristics, except that LRV produces

TABLE 3.—*Evidence of a "latent" period within the vector Aphis gossypii as essential for transmission of lily-rosette virus, lily-symptomless virus, and the necrotic-fleck complex from Easter lily to Easter lily*

Latent period (days)[a]	Virus or complex transmitted								
	Lily-rosette virus			Lily-symptomless virus			Necrotic-fleck complex		
	Total tests	Positive tests	Plants affected[b]	Total tests	Positive tests	Plants affected[b]	Total tests	Positive tests	Plants affected[b]
2	...	...	.....	1	0	0/10	1	0	0/10
3	1	0	0/10	...	...	.....	3	0	0/21
4	...	...	.....	...	...	.....	3	0	0/13
6	...	...	.....	2	0	0/16	...	...	.....
7	...	...	.....	...	...	.....	1	1	2/5
8	2	1	1/12	1	0	0/14	2	2	9/10
9	...	...	.....	...	...	.....	2	1	3/10
10	1	1	10/10	...	...	.....	1	1	3/10
Over 10	6	6	35/38	13	10	47/125	25	22	120/217

[a] Total elapsed time of feeding on both source plant and test seedling.

[b] Number of plants affected over number exposed. The transmission of lily-symptomless virus was determined by mechanical addition of cucumber-mosaic virus to the test seedlings either before or after these were exposed to feeding of *Aphis gossypii* from Norwood Crole lilies.

well-marked symptoms in the Easter lily while LSV produces none. Easter lilies carrying the symptomless virus, however, are not protected against infection by lily-rosette virus (Fig. 9).

In trials with *Aphis gossypii* conducted under apparently favorable conditions 93.8 per cent of 48 plants were infected with LRV, 54.7 per cent of 247 plants with the NF complex, and 37.6 per cent of 125 plants with LSV. The lily-rosette virus, apparently a single entity, is carried with much higher efficiency than is the NF complex. The low percentage transfer of LSV alone presumably indicates that the test for its presence, namely mechanical addition of CV, is not always highly efficient.

It was noted in some of our earliest trials that the mottle virus (LMV) could not be recovered from many of the Easter lily seedlings typically flecked as a result of *Aphis gossypii* transmission. Again in a recent series

TABLE 4.—*Evidence of the retention of lily-rosette virus and of lily-symptomless virus by Aphis gossypii, when first fed on Easter lily source plants, then on non-susceptible plants, and finally on Easter lily test seedlings*

Interval on non-susceptible plants		Retention of virus as shown by final feeding on Easter lily seedings[a]
Species	Days	
Lily-rosette virus		
Chrysanthemum	3	5/5
Tulip	7	3/7
Tulip and Chrysanthemum	7 } 2 }	5/5
Lily-symptomless virus		
Chrysanthemum	3	4/10[b]

[a] Plants affected over number exposed.
[b] Determined by expression of necrotic-fleck symptoms after addition of cucumber-mosaic virus.

of subinoculations LMV was detected in only 2 of 10 typical examples of necrotic fleck induced by this vector. Similarly, lily-rosette virus as received from Florida in commercial Easter lilies was found to be accompanied by LMV, but transfers to seedlings by *A. gossypii* were usually free from LMV. Evidently LMV is not essential for expression of necrotic fleck symptoms (Fig. 10), or for expression of rosette symptoms (Fig. 11), although it has been found generally present in commercial Easter lilies affected with necrotic fleck or rosette. Evidence presented elsewhere (4) shows that *A. gossypii* is a relatively inefficient vector of LMV. This vector, therefore, offers a means of separating LSV or LRV from the LMV that commonly accompanies them in commercial Easter lilies. Preliminary evidence recently gained indicates that these separations are highly efficient when a part of the latent period in *A. gossypii* is caused to elapse while the vector feeds on a plant such as chrysanthemum that is non-susceptible to LMV.

ANALYSIS AND SYNTHESIS OF NECROTIC FLECK IN EASTER LILIES

Of the three viruses (CV, LSV, LMV) commonly present in commercial samples of necrotic fleck, CV is readily separable by passage through tobacco,

as the other 2 viruses fail to infect this plant. LMV has been separated from the CV and LSV by mechanical inoculation to tulip and thence to lily. When this separation was first reported (1), tulips were not known to be susceptible to CV, and tulip was believed to be a differentiating host. It was

FIG. 10. Symptoms of necrotic fleck in Easter lily seedling induced by *Aphis gossypii*. Subinoculations show lily-mottle virus absent. Photographed (in 5-inch pot) 2 years after exposure.

subsequently found (3) that tulip is susceptible to CV. The fact that this separation succeeded may be attributed to chance failure of CV in the inoculation to or from tulip, or to more rapid movement of LMV in tulip. No better means of separating LMV from CV is known, as all plants known to be

FIG. 11. Symptoms of lily rosette (yellow-flat) induced in Easter lily seedling by *Aphis gossypii*. Subinoculations show lily-mottle virus absent. Photographed (in 5-inch pot) 34 days after exposure.

susceptible to the former are also susceptible to CV, and the properties of these 2 viruses are similar. Separation of LSV from the others is accomplished by *Aphis gossypii* under certain conditions, as discussed above.

In practice the separation of LMV and LSV from CV is not necessary, as these two occur in the absence of CV in certain commercial stocks of Easter lilies that show no fleck symptoms. We have made use of the "Norwood" clonal line of Creole Easter lily, first selected and propagated at Louisiana State University by Dr. Fred Cochran, and further increased and maintained as a known stock at the Plant Industry Station, Beltsville, Md. This usually symptomless clon yields LMV on mechanical transfer and LSV (rarely LMV

TABLE 5.—*Synthesis of necrotic fleck in Easter lily seedlings by sap transfer of cucumber-mosaic virus followed by Aphis gossypii transfer of lily-symptomless virus*

Cucumber-mosaic virus, by sap			Lily-symptomless virus, by *Aphis gossypii*			Necrotic fleck developing in	
Source	Date	Plants inoc.	Source	Date	Plants inoc.	Inoc. plants[a]	Control plants[a]
	(1940)			(1941)			
Easter lily	Feb. 8	24	Norwood Easter lily	Feb. 21	10	8/10	0/14
Tobacco	Dec. 18	25	"	Mar. 12	10	10/10	0/15
				(1942)			
Tobacco	Dec. 18	10	"	Jan. 29	5	4/5	0/5

[a] Expressed as number of plants infected over number treated.

also) on transfer by *Aphis gossypii*. The presence of LSV was first postulated from the fact that this clon yields fleck on addition of CV while seedlings do not (Table 1).

In the experiments summarized in table 5, CV + LMV were first established in Easter lily seedlings as a test of the capacity of these 2 viruses to induce NF. No NF symptoms appeared, as already indicated (Table 1). After 3 to 13 months' incubation, the plants in each test were divided into 2 lots, 1 lot then being exposed to *Aphis gossypii* from Norwood Creole Easter lilies known to carry LSV and LMV. Twenty-two of the 25 plants exposed to infection by both CV and LSV developed necrotic fleck symptoms 33 to 37 days after the second inoculation (Figs. 2, 4), while 34 controls showed no fleck. CV was found present in 16 of 18 control plants indexed in 1943. It is evident from these experiments that a symptomless component LSV essential for necrotic fleck was present in the Norwood Easter lilies, and that this component was carried by *A. gossypii*.

Because of the design of these particular tests, LMV was also present in the fleck synthesized. It should be equally feasible to synthesize necrotic fleck from the 2 essential components (LSV + CV) without LMV. This has been accomplished in a number of trials, but the percentage of necrotic fleck resulting has been low. Low proportions of necrotic fleck in attempted syntheses are considered due to low virulence of CV in some inocula, and also to peculiarities in the feeding habits and virus relations of the vector *Aphis gossypii*, as has been discussed above.

It is considered feasible to produce pure cultures of LSV in Easter lily seedlings by the selective transfer of this virus from Norwood Easter lilies by *Aphis gossypii*. Unfortunately the only available test for the presence of LSV is to add CV, the production of fleck symptoms signifying the presence of LSV. If aphid transfers are made in 1 season and test inoculations in the following season after the bulbs have been divided, it is possible to establish the presence of LSV by inducing NF in 1 division of a test plant while retaining another division as a pure culture of LSV.

In one experiment necrotic fleck was synthesized by two species of aphids, *Myzus persicae* transmitting CV and *Aphis gossypii* transmitting LSV. Nine Easter lily seedlings were first exposed to *M. persicae* from a Creole lily showing NF symptoms (CV + LSV + LMV), with the resulting appearance of mottling (CV + LMV) but no flecking. Later *A. gossypii* was transferred from Norwood Creole (LSV + LMV) to these 9 seedlings. Four of the 9 plants developed necrotic fleck symptoms (CV + LSV). This experiment would suggest the possibility of spread of necrotic fleck by *M. persicae* in commercial fields of Easter lilies (LSV + LMV), in which LSV is general and CV is occasional in necrotic flecked individuals (CV + LSV + LMV). In such lily fields there is every reason to expect spread of CV with the resultant appearance of necrotic fleck, even in the absence of the vector *A. gossypii*, for *M. persicae* and other vectors can transfer CV, the only constituent that is lacking. If such vectors of CV move across lily fields when migrating from

other crops, rapid dissemination of CV comparable to that noted for LMV (4) may be expected.

HOST RANGE OF NECROTIC FLECK

As indicated in table 2, only Easter lily is established as a suscept of both constituent viruses of necrotic fleck (CV and LSV), while the susceptibility of tulip, *Colchicum*, and *Gloriosa* to LSV is left in doubt. Early in the present study a number of different plant species, chiefly Monocots, were exposed to necrotic fleck inoculation by *Aphis gossypii*. It was believed at the time that such tests might provide evidence on host relations of all 3 viruses (CV, LSV, LMV) present in flecked commercial Easter lilies. It was learned subsequently that this vector carries LMV infrequently, and also that it may carry both CV and LSV to some plants, and one or the other of these separately to others. The customary disclaimer as to significance of negative evidence is therefore appropriate at this point.

Inoculations to Easter lily seedlings were made parallel with tests on blocks of other possible suscepts, and most of these indicated valid test conditions. Subinoculations to detect CV or LMV were made from the majority of the species exposed, and from all that showed symptoms. No evidence was found in these trials that any of the species enumerated below are susceptible to any of the 3 viruses in question, the positive and possibly significant results having been summarized above (Table 2):—

AMARYLLIDACEAE—*Vallota purpurea* Herb.; ARACEAE—*Philodendron* sp.; COMMELINACEAE—*Commelina coelestis* Willd., *C. nudiflora* L.; CRUCIFERAE—*Brassica rapa* L. (turnip); DIOSCOREACEAE—*Dioscorea alata* L.; IRIDACEAE—*Belamcanda chinensis* (L.) DC.; *Freesia hybrida* Hort., *Iris filifolia* Boiss. (var. Imperator), *I. pallida* Lam., *I. versicolor* L., *Moraea iridioides* L., *Tigridia pavonia* (L. f.) Ker-Gawl., *Tritonia crocata* (L.) Ker-Gawl.; LILIACEAE—*Agapanthus africanus* (L.) Hoffmg., *Allium cepa* L. (var. Yellow Globe), *A. cernuum* Roth, *A. odorum* L., *A. speciosum* Cyr., *Aloe* sp., *Asparagus asparagoides* (L.) W. F. Wight, *A. sprengeri* Regel, *Asphodeline lutea* (L.) Reichb., *Brodiaea uniflora* (Lindl.) Engl., *Camassia leichtlinii* (Baker) S. Wats., *Convallaria majalis* L., *Dracaena sanderiana* Sander, *Erythronium* sp., *Galtonia candicans* Decne., *Haworthia altilinea* Haw., *Hosta plantaginea* Aschers., *Kniphofia tucki* Baker, *Lilium hansoni* Leichtl., *L. henryi* Baker, *L. humboldtii* Roezl & Leichtl., *L. pardalinum* Kellogg, *L. parryi* S. Wats., *L. parvum* Kellogg, *L. sargentiae* Wilson, *L. superbum* L., *Medeola virginica* L., *Muscari polyanthum* Boiss., *Nothoscordum fragrans* (Vent.) Kunth, *Ophiopogon jaburan* (Siebold) Lodd., *Ornithogalum thyrsoides* Jacq., *Sansevieria zeylanica* Willd., *Smilacina racemosa* (L.) Desf., *Smilax* sp., *Tricyrtis hirta* Hook., *Trillium* sp., *Uvularia sessilifolia* L., *Yucca baccata* Torr., *Y. flaccida* Haw.; MUSACEAE—*Musa cavendishii* Lamb., *M. textilis* Née; ZINGIBERACEAE—*Hedychium coronarium* Koen.

Failure to produce CV infection in *Commelina nudiflora* and *Musa cavendishii* is surprising in view of Wellman's (21) success in producing symptoms in both these plants with the celery strain of CV on transfer of *Aphis gossypii*. Inasmuch as parallel trials on Easter lily seedlings were successful, the above results indicate that *Commelina* and *Musa* are not readily affected by lily strains of CV. Natural infection has not been detected in *C. nudiflora* growing as a weed near flecked Easter lilies in Louisiana, nor in bananas occasionally grown for ornament in that State. The commonly heavy infestations of *A. gossypii* on flecked Creole lilies in some sections of Louisiana should produce ample opportunity for infection of the *Commelina* growing adjacent. This weed is reported commonly affected with the celery strain of CV in celery districts of Florida, and is considered an important reservoir of the virus (7).

Lilium sargentiae and *L. superbum*, though not infected in these tests, have been found naturally infected with CV and with LMV (2). It is to be expected that *Aphis gossypii* will carry CV to these and other species of lilies under some conditions, and other vectors of CV, *Macrosiphum solanifolii* and *Myzus persicae*, may also do so.

DESCRIPTION OF LILY-SYMPTOMLESS VIRUS

The evidence developed above is believed adequate to establish the existence of a symptomless constituent of necrotic fleck. This evidence may be summarized as follows: (1) Analysis of naturally occurring necrotic fleck reveals cucumber-mosaic virus (CV) and mottle virus (LMV), but analysis of some experimentally produced necrotic fleck reveals only CV. LMV is, therefore, not an essential constituent of fleck. (2) CV does not produce necrotic fleck in previously virus-free Easter lilies, but does so in certain symptomless commercial Easter lilies. (3) Mechanical transfer from such symptomless Easter lilies to seedlings experimentally infected with CV does not produce necrotic fleck symptoms, but *Aphis gossypii* transfer from the same symptomless source plants does produce fleck. (4) The necrotic-fleck complex has a specific vector, *A. gossypii*, and a well-defined latent period in this vector, whereas CV has several vectors with no such latent period in these.

The symptomless virus LSV, which, in combination with CV, produces necrotic fleck in Easter lilies, does not fall into any of the categories thus far erected for plant viruses. Although the possibility always remains that symptoms may be produced by this virus in some other plant, the tests thus far conducted have demonstrated only one host plant, *Lilium longiflorum*. Inasmuch as this virus is transmitted by an aphid, *Aphis gossypii*, and with difficulty by sap, it shows affinity with the group of viruses placed in *Marmor* (11), and could be placed in the family Marmoraceae if the concept of this family could be broadened to include symptomless forms. Lily-symptomless virus, therefore, is made the type of a new genus *Adelonosus*, which is tentatively assigned to Marmoraceae.

Adelonosus (new genus): Adelos (invisible) + nosos (disease).[3]

Viruses capable of multiplying in living plants, but producing no recognizable symptoms in these, except on interaction with distinct viruses with which they form complexes. Transmitted by aphids, by sap, or by both means.

Type species *Adelonosus lilii*.

Adelonosus lilii (n. sp.).

Common name: Lily-symptomless virus.

Geographic distribution. United States, Japan. Probably coextensive with commercial culture of Easter lily (*Lilium longiflorum*).

Host range: LILIACEAE—*Lilium longiflorum*. No other host established.

Symptoms: No symptoms in pure culture in Easter lily seedlings; necrotic fleck when present together with cucumber-mosaic virus (*Marmor cucumeris* Holmes).

Methods of transfer: By sap with difficulty (3.6 per cent transfer of the complex *Adelonosus lilii* + *Marmor cucumeris*). By *Aphis gossypii* only, among the insect species tested; with a latent period of a few days in this vector. Not through seed.

Properties: Not determined.

Test reaction: Addition of *Adelonosus lilii* to Easter lilies induces necrotic flecking when cucumber-mosaic virus is also present.

DISCUSSION

The conception of a completely symptomless virus is advanced with some reluctance by the writers. It is recognized that a single test plant expressing symptoms would reduce the proposed genus *Adelonosus* to synonymy, possibly with *Marmor*. Such a test species would be of very great value in studies of distribution and properties of this virus. A fairly extensive search for such a test species has therefore been made. Further search will be justifiable if the techniques of manipulating this virus and its vector can be appreciably improved.

The proposed common name, lily-symptomless virus, is acknowledged to be awkward, but the more fitting name, lily-latent virus, has been preempted by McWhorter (13) for a member of the mottle or tulip-breaking group (4), to which Holmes (11) has assigned the name *Marmor mite*.

Lily-symptomless virus (LSV) is far more common in Easter lilies than is cucumber-mosaic virus (CV). This is a surprising fact inasmuch as LSV is known to occur only in Easter lilies and is transmitted in nature only by the melon aphid, whereas CV occurs in many other plants and is transmitted by several other vectors. A smaller discrepancy in range and prevalence might be accounted for by more efficient transfer of LSV by *Aphis gossypii*, as was indicated in some experimental transfers of the necrotic fleck complex, and by evidence that LSV is persistent in its vector. However, the wide difference in prevalence noted suggests that LSV has long been common in most Easter lily varieties, and that CV has more recently become prevalent in these.

The Creole Easter lilies grown for generations in the Delta district of Louisiana are said to have been free from necrotic fleck until recent years, the appearance of the fleck disease dating from the introduction of other Easter lily varieties (Giganteum, Harrisi) for comparison. As late as 1941, when necrotic fleck was prevalent near these centers of introduction, this disease was still absent from isolated plantings of Creole lilies in other Louisiana parishes as well as in Alabama, Florida, South Carolina, and Virginia. The

[3] The writers are indebted to Charles Drechsler for selection of the generic name.

origin of the Creole variety is not known, but it seems probable that lily-symptomless virus was introduced and disseminated with this variety, and that the necrotic fleck disease appeared later after a second separate introduction.

Similarly the Croft and other varieties of Easter lily long cultivated in Oregon are remarkably free of the necrotic fleck disease, but samples tested have proved to be carriers of lily-symptomless virus. The origin of these varieties also is in doubt, but a recent seed origin does not readily account for the prevalence of this virus unless the seedlings were at some time grown near affected Easter lily varieties in a section where *Aphis gossypii* was prevalent.

The fact that Easter lily varieties such as Creole and Croft have, in certain sections, remained free from necrotic fleck for years, shows that cucumber-mosaic virus does not commonly spread from other plants to lilies, inasmuch as only CV is needed to produce necrotic fleck in Easter lilies already carrying LSV. As shown above, the necrotic-fleck complex is readily produced by mechanical addition of CV to such Easter lilies. It may be assumed that any vector of CV that migrates from plants affected with CV can thus produce fleck in such commercial lilies. In other words, though *Aphis gossypii* is the only true vector of the necrotic fleck complex (CV + LSV), any vector of CV might appear to be a vector of necrotic fleck to commercial stocks already affected with LSV. As evidence of such natural synthesis of necrotic fleck is rarely if ever detected in commercial Easter lily culture, the indication is either (1) that there is little natural migration of vectors from other reservoirs of CV to lilies, or (2) that such reservoirs of CV are uncommon in districts of commercial Easter lily culture.

Aphis gossypii is the principal vector of CV in cucurbits (6) as well as in lilies. In our tests this aphid has not been readily transferred from cucumber to Easter lily or vice versa, and presumably does not migrate readily from one to the other in nature. Similarly, free migration from *Echinocystis lobata* Torr. & Gray, the wild cucumber in which this virus is seed-borne, is not to be expected. No information is at hand concerning the freedom of movement of this or other aphid vectors from lily to the recognized perennial hosts of CV, namely *Asclepias syriaca* L., *Phytolacca decandra* L., *Physalis heterophylla* Nees, *P. subglabrata* Mackenzie and Bush, *Nepeta cataria* L. It may be further noted that these potential reservoirs of CV have been found affected only in the vicinity of cucurbit cultures, and that they have not been detected as carriers of CV near Easter lily cultures. It therefore appears, in spite of other possibilities mentioned, that commercial Easter lilies become infected with necrotic fleck chiefly, if not exclusively, from other infected Easter lilies.

Easter lilies produced from seed, and suitably isolated from bulb-perpetuated lilies, remain virus-free. Evidence against seed transmission of CV and LMV is presented elsewhere (4), while evidence against seed carriage of LSV is afforded by data such as those in Sect. A of table 1. Inas-

much as mottle viruses (4) and LSV have very restricted host ranges and, as indicated above, CV does not appear to spread readily to lilies from other sources, there is reason to believe that adequately isolated seedling Easter lilies will remain virus-free indefinitely. Encouraging as they are, these findings are of technical interest only. Present and prospective American producers of Easter lilies are agreed that virus-free culture is impractical. The facts that Easter lily seedlings are too variable for immediate use in forcing, and that rigid isolation must be practiced during the years of selection and increase of new varieties, remove such an undertaking from the field of the commercial producer.

Demonstration of the separate etiology of mottle and necrotic fleck is, on the other hand, of value to such producers who have for practical reasons decided to rogue necrotic fleck but to ignore mottle. As mottle is not an essential constituent of necrotic fleck, mottling can logically be evaluated as a separate disease.

SUMMARY

The necrotic fleck disease of Easter lilies is described, and 2 sub-types, sparse fleck and very sparse fleck, are distinguished in symptom expression, but not in etiology.

The necrotic-fleck virus complex is mechanically transmissible to Easter lily seedlings with difficulty (about 4 per cent successful), but is readily transferred (87 per cent) to commercial Easter lilies.

Cucumber-mosaic virus does not induce necrotic fleck symptoms in previously virus-free Easter lily seedlings, but does induce these symptoms in commercial Easter lilies.

Cucumber-mosaic virus and lily-mottle virus together fail to produce necrotic fleck in previously virus-free Easter lily seedlings.

Only *Aphis gossypii* has been found to transmit necrotic fleck to previously virus-free Easter lily seedlings. *Macrosiphum solanifolii* and *Myzus persicae* have been found to carry cucumber-mosaic virus and lily-mottle virus, but not fleck, to such seedlings. *Aphis fabae, Macrosiphum lilii, Myzus circumflexus,* and *M. convolvuli* were not found to carry any virus from necrotic-flecked Easter lilies to seedlings.

Aphis gossypii transmitted cucumber-mosaic virus from necrotic-flecked Easter lilies to *Calochortus* sp., *Colchicum autumnale, Gloriosa rothschildiana,* and *Fritillaria pudica,* not previously recorded as being susceptible to this virus.

Lily-mottle virus, which commonly accompanies necrotic fleck in Easter lilies, has been found absent from some experimentally produced necrotic fleck in Easter lily, and is therefore not an essential component of necrotic fleck.

A lily-symptomless virus is postulated as an essential component of necrotic fleck, producing necrotic fleck symptoms when present with cucumber-mosaic virus.

Lily-symptomless virus is apparently confined to Easter lily, but the susceptibility of tulip, *Colchicum*, and *Gloriosa*, to this virus remains in doubt. No evidence of susceptibility to this virus was found in other plants tested, namely 56 species representing 40 genera and 9 families, chiefly Monocotyledons.

Lily-symptomless virus occurs in symptomless Creole Easter lilies known as the Norwood strain, in mixture with lily-mottle virus. Sap transfers from the Norwood strain do not produce necrotic fleck in Easter lily seedlings previously infected with cucumber-mosaic virus. Transfers from the same Norwood source by *Aphis gossypii* do produce fleck in such seedlings, thus synthesizing necrotic fleck. Transfers of cucumber-mosaic virus to Norwood Creole, either by sap or by the vector, readily synthesize necrotic fleck.

Lily-symptomless virus passes a latent period of some 4 to 6 days in *Aphis gossypii*. This permits separation of this virus from lily-mottle virus.

A new genus, *Adelonosus*, is created for plant viruses inducing no symptoms, and *A. lilii*, the lily-symptomless virus, is named as the type species.

The distribution and economic significance of necrotic fleck and of the symptomless component are discussed.

PLANT INDUSTRY STATION,
BELTSVILLE, MD.

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BLACK SCALE: A DISEASE OF EASTER LILY BULBS[1]

A. G. PLAKIDAS

(Accepted for publication January 10, 1944)

In August, 1937, a specimen of fresh-dug bulbs of easter lily (*Lilium longiflorum*) showing dark-brown to black lesions was brought to this laboratory by D. L. Gill, who collected it in a field in Boothville, Louisiana. A second lot of diseased bulbs from Buras, Louisiana, was brought in a few days later by F. D. Cochran. Both men reported the disease as serious in many lily fields in Plaquemines Parish, and causing considerable concern to the growers.

In October of the same year, the writer visited the lily-growing district to obtain additional material for study and more information on the distribution and importance of the disease. The growers differed in their opinions as to the cause of the disease, some blaming it on poor drainage, others on drought, and others on the fertilizer used, but there was complete unanimity among them that this apparently new disease was extremely destructive, and that it threatened the commercial growing of lily bulbs. Losses as high as 80 per cent were reported by several of the growers. One of the oldest lily growers in the area reported that of a total of 40,000 bulbs he harvested in 1937, only about 7,000 were salable; the rest were diseased.

All the growers interviewed appeared to agree that the disease was new to their area. They stated that a few diseased bulbs were observed in some fields 3–4 years earlier, but that serious losses were not incurred prior to about 1936. Granted that reliance can be placed on these statements of the growers, it is of interest to speculate upon where the disease came from. It is known that on more than one occasion lily bulbs from Bermuda, where the disease is known to occur, have been planted in the lily-growing area of Louisiana, and it may be inferred that the disease was introduced on these bulbs. This, however, is only a speculation.

A study of the disease was undertaken in 1937 and continued for two years. The information obtained from this study showed conclusively that the black scale is caused by a fungus belonging to the genus *Colletotrichum*. Except for a brief abstract (5), publication of these findings has been delayed in the hope that further work might be done on the problem and particularly on efforts to devise some effective control measure. As resumption of the investigations does not appear feasible at present, it has seemed advisable to publish the information obtained with the hope that it may be useful to other investigators.

[1] The disease is known locally under various names, such as "black bulb," "brown bulb," "brown scale," or "black scale." In a preliminary report (5) the writer used the term "brown scale." The name "black scale" has priority, since it has been used to designate what appears to be the same disease on Easter lily bulbs in Bermuda (2, 3, 7, 8), and, for the sake of uniformity, will be used in this publication.

DESCRIPTION OF THE DISEASE

Macroscopic Symptoms. Freshly dug normal bulbs are white to lemon-yellow. In contrast, diseased bulbs show varying degrees of color from brown to nearly black, depending on the number and size of the lesions on the scales (Fig. 1, A). The injury extends to several layers of the scales, the outermost being the most severely affected. Young lesions appear as more or less irregular light-brown areas on the scales. At this stage, the

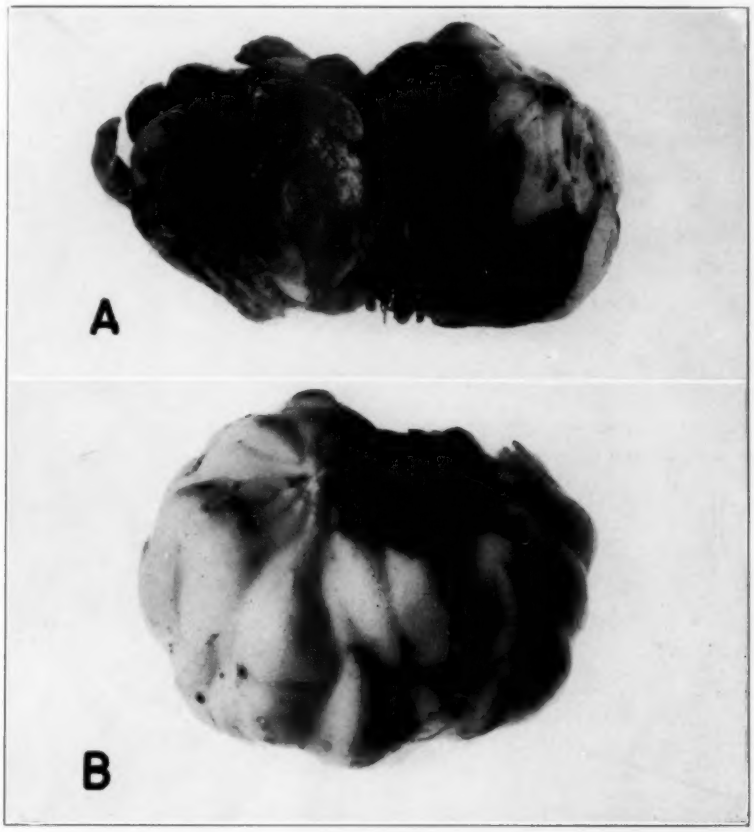

FIG. 1. Black scale of Easter lily. A. Severely diseased bulb. B. Healthy bulb. Approximately natural size.

injury is limited to the epidermal cells. Older lesions are darker-brown and somewhat sunken, due to collapse of the epidermis and one to two layers of the subepidermal cells. Still older lesions are nearly black, and the affected tissues are shrivelled and dried. The lesions are more numerous on the apex half of the scales and, for the most part, occur on the outer (convex) surface, though often are found also on the inner (concave) surface. The plates (stems) and roots appear not to be affected. The core even of severely diseased bulbs appears perfectly normal, and it is known that when diseased

bulbs are planted they grow almost normally and produce flowers. However, because of their unsightly appearance, diseased bulbs are unsalable.

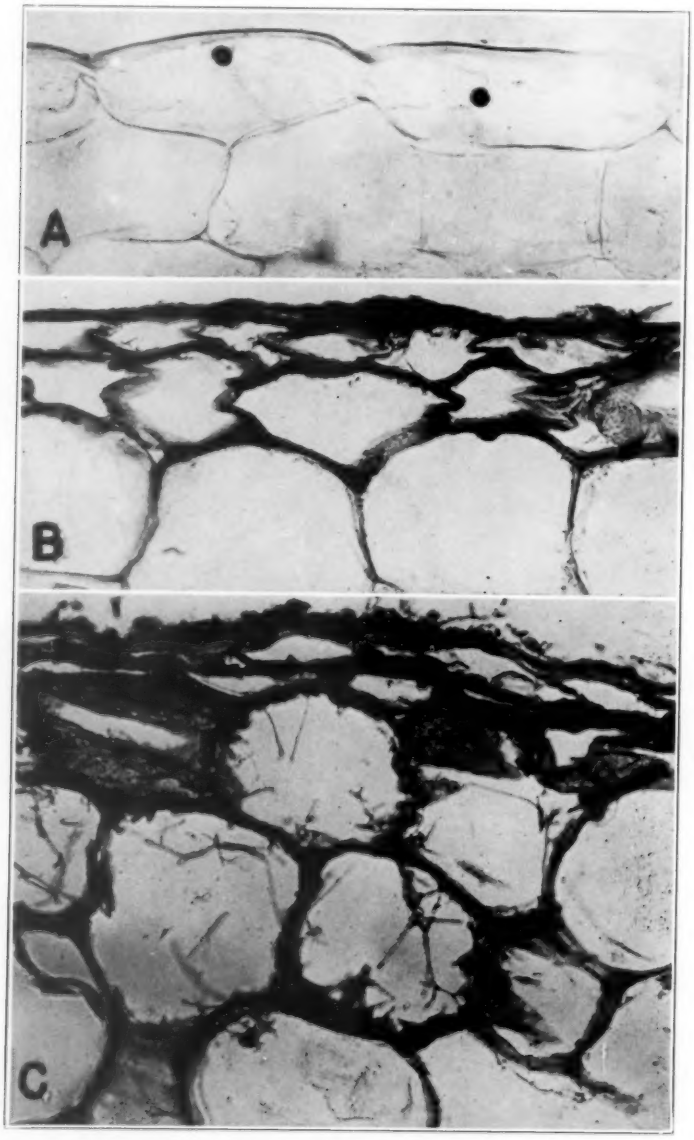

FIG. 2. A. Section through healthy bulb scale showing normal epidermal and first layer of subepidermal cells. B. Section through young lesion showing injury to the epidermis and the first layer of subepidermal cells. C. Section through an older lesion showing injury extending to the fourth subepidermal layer of cortical cells. The epidermis and the first layer of cortical cells have collapsed, and the fungus invasion extends to the fourth layer of subepidermal cells. All $\times 200$.

Microscopic Symptoms: Pathological Histology. The description of the microscopic symptoms is based on examination of hundreds of sections (both

free-hand and paraffin preparations) of diseased scales made through lesions in different stages of development.

In the very young lesions (light-brown spots) the epidermal and the first layer of subepidermal cells are definitely injured, as shown by their brown discoloration, their somewhat flattened shape, and the thickening and flabbiness of their walls (Fig. 2, A, B). At this stage, mycelium occurs sparingly in the tissue, and consists of rather thick hyphae of irregular diameter, occurring between the epidermal and the first layer of subepidermal cells. Mycelium is present on the surface of the lesions at this stage, and it is probable that the fungus kills or injures the tissue in advance of invasion. In later stages (lesions dark-brown and somewhat sunken) the epidermis and the first layer of subepidermal cells are completely collapsed, and mycelium now occurs very abundantly on the surface, beneath the cuticle, and inside the epidermal and first layer of subepidermal cells, and progressively less abundantly in the second, third, and fourth subepidermal layers of cortical cells (Fig. 2, C). No attempt was made to study the disease in old, shrivelled scales because these were usually invaded by secondary organisms, particularly *Fusarium* and *Penicillium*. In general, it appears that in young and moderately old lesions the injury caused by *Colletotrichum* is relatively shallow, limited to the epidermis and the first 2 to 4 layers of subepidermal cells.

FUNGI ASSOCIATED WITH THE DISEASE

Three fungi, species of *Colletotrichum*, *Fusarium*, and *Penicillium*, were those most commonly isolated when tissue plantings were made from scale lesions. The last-named fungus resembles *P. cyclopium* Westling (4) in culture, but no attempt was made to identify it definitely. It appeared that the longer the bulbs were kept in storage the greater was the percentage of *Fusarium* and *Penicillium* cultures obtained from the tissue plantings and the harder it was to isolate the *Colletotrichum*. On the other hand, when tissue plantings from freshly dug bulbs were made, *Colletotrichum* was isolated very readily and almost exclusively. For example, out of 37 colonies developing from tissue plantings from a specimen of freshly dug bulbs on August 15, 1943, 30 were *Colletotrichum*, 4 *Fusarium*, and 3 unidentified. From another lot of bulbs, which had been in storage for several months, out of 28 colonies growing out of tissue plantings 16 were *Fusarium*, 8 *Peni-*

TABLE 1.—*Fungi isolated from tissue plantings from black scale lesions*

Lot	No. of isolations	*Fusarium*	*Colletotrichum*	*Penicillium*	Other fungi
1	179	60	71	48	0
2	27	12	12	1	2
3	85	31	28	25	1
4	37	4	30	0	3
5	40	28	4	8	0
6	62	14	33	10	5
Total	430	149	178	92	11

cillium, and 4 *Colletotrichum*. The isolations made from 6 different lots of diseased bulbs are summarized in table 1.

PATHOGENICITY

Laboratory Tests

As a preliminary test, the pathogenicity of the fungi isolated was studied in the laboratory. Healthy scales and bulblets were surface-sterilized with 1–1000 $HgCl_2$, washed in sterile water, dried on sterilized filter paper, placed in moist chambers, and inoculated with various isolates of *Fusarium*, *Penicillium*, and *Colletotrichum* and with combinations of the 3 fungi. The inoculum was placed on punctures made with a flamed needle.

Results. Rotting was caused by all 3 fungi, the *Fusarium* isolates being the most aggressive and the *Penicillium* isolates the least. *Fusarium* and *Penicillium* produced a soft type of rot. In only two instances did *Colletotrichum* isolates produce a more or less superficial, slightly sunken, dry type of lesions, which resembled somewhat the lesions as they occur naturally in the field. In other instances the rot was soft; deep-seated, not much different from that produced by *Fusarium* and *Penicillium*.

Soil Infestation Tests

It was evident that the laboratory method of testing the pathogenicity of these fungi was not satisfactory. In subsequent experiments, the 3 fungi were used, alone or in combination, to infest soil in which healthy bulbs were planted. This method, which approximated field conditions, furnished conclusive evidence that the *Colletotrichum* is the cause of the black scale disease.

Experiment 1. Soil was taken from a field from which about 80 per cent of the bulbs harvested in 1937 were diseased. The soil was divided into two lots, one of which was sterilized by autoclaving for 4 hours; the other was left non-sterilized. Each lot was then subdivided into 12 series. The scheme of the experiment and the results obtained are shown in table 2.

The inoculum was prepared by growing the respective fungi in Mason jars on autoclaved grain (3 parts oats, 1 part wheat). The inoculum was mixed with the soil in the soil-infestation series.

The cottonseed meal (Series C, D, O, and P) was added to the soil in order to test the claims of some of the growers that the disease was either caused by cottonseed meal, or was much more severe when cottonseed meal was used as fertilizer.

The duplicate series (poor drainage), in which glazed porcelain crocks instead of pots were used as containers, were included in order to determine the effect of poor drainage. Several of the growers maintained that the disease was much more severe on poorly drained land. The crocks were not perforated, and the soil in them was flooded 3 times during the course of the experiment to simulate poor drainage conditions. In the "good drainage" series, 7-inch perforated pots were used as containers.

It will be noted by examining the arrangement of the experiment, that the *Fusarium* and *Colletotrichum* inocula (series G, H, S, and T) were used in combination instead of separately, as was done with *Penicillium*. There were several reasons for this. Firstly, the amount of sterilized soil and the number of healthy bulbs at hand when the experiment was being set up were limited and did not allow inclusion of additional series. Secondly, it was suspected that *Penicillium* was the causal organism; so care was taken to use this fungus separately.

TABLE 2.—*Pathogenicity studies. Arrangement and results of experiment 1*

Series and treatment	No. bulbs harvested[a]	No. bulbs diseased	Per cent diseased
Lot I. Non-sterilized soil			
A. Check; non-inoculated, good drainage	7	3	42.8
B. Check; non-inoculated, poor drainage	6	0	0.0
C. Cottonseed meal; good drainage	11	5	45.4
D. Cottonseed meal; poor drainage	8	2	25.0
E. *Penicillium;* good drainage	11	4	36.4
F. *Penicillium;* poor drainage	10	4	40.0
G. *Fusarium* and *Colletotrichum;* good drainage	17	13	76.5
H. *Fusarium* and *Colletotrichum;* poor drainage	7	6	85.7
I. *Fusarium, Colletotrichum* and *Penicillium;* good drainage	17	17	100.0
J. *Fusarium, Colletotrichum* and *Penicillium;* poor drainage	14	11	78.6
K. Diseased bulbs; good drainage	11	11	100.0
L. Diseased bulbs; poor drainage	19	19	100.0
Lot II. Sterilized soil			
M. Check; non-inoculated soil; good drainage	10	1	10.0
N. Check; non-inoculated soil; poor drainage	13	0	0.0
O. Cottonseed meal; good drainage	10	0	0.0
P. Cottonseed meal; poor drainage	2	0	0.0
Q. *Penicillium;* good drainage	9	1	11.1
R. *Penicillium;* poor drainage	15	1	6.6
S. *Fusarium* and *Colletotrichum;* good drainage	13	13	100.0
T. *Fusarium* and *Colletotrichum;* poor drainage	4	1	25.0
U. *Fusarium, Colletotrichum* and *Penicillium;* good drainage	20	20	100.0
V. *Fusarium, Colletotrichum* and *Penicillium;* poor drainage	1	1	100.0
W. Diseased bulbs; good drainage	17	17	100.0
X. Diseased bulbs; poor drainage	7	7	100.0

[a] The number of bulbs harvested was not the same as the number planted because in most cases they multiplied, while in some cases in the poor drainage series soft rot set in and rotted completely some of the bulbs.

The diseased bulbs (series K, L, W, and X) were included in order to see what effect the disease had on their growth and to determine whether or not they would recover. All the other series were planted with healthy bulbs.

The experiment, which was carried on in the greenhouse, was started on October 14, 1937, and the bulbs harvested and examined on June 13, 1938.

Results. The results of experiment I are summarized in table 2. Examining first the first half of the table (Lot I, non-sterilized soil), we see that the disease occurred in all series (except in series B, poor drainage; the

effect of poor drainage is discussed below). This was to be expected, for, as stated in the foregoing, the soil used for the experiment came from a field in which the disease was very severe. Fragments of diseased scales were present, so the soil was well-contaminated. However, even in non-sterilized soil, the series in which *Fusarium* and *Colletotrichum* were used as inoculum (G, H, I, and J) showed a much higher percentage of disease than either the

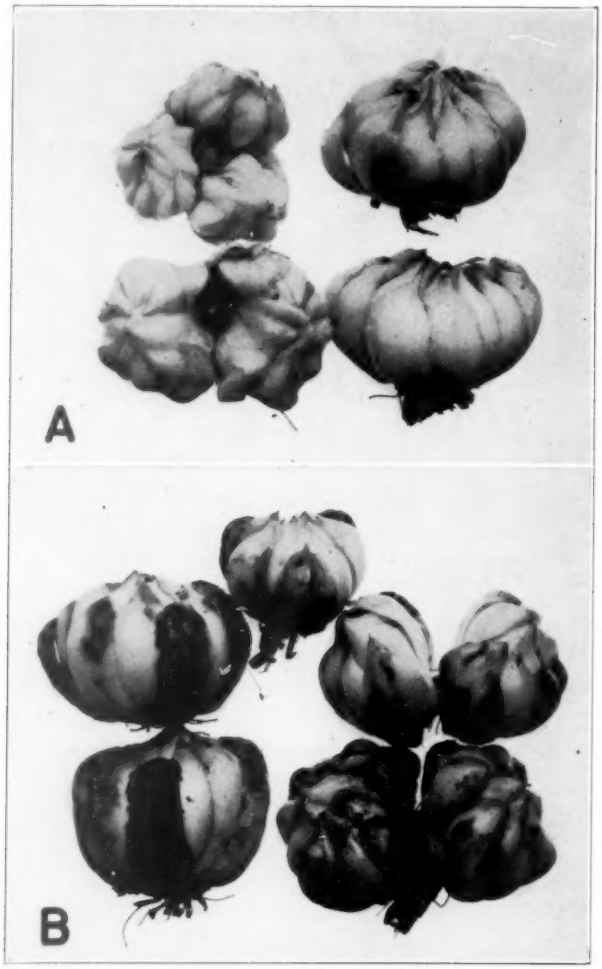

FIG. 3. A. Check. Healthy bulbs grown in sterilized non-inoculated soil. B. Typical black scale symptoms on bulbs grown in soil inoculated with *Colletotrichum lilii* and *Fusarium* spp. Approximately ½ natural size.

check or the *Penicillium* series. The second half of the table (Lot II, sterilized soil) shows definite and clear-cut results. Here, in all series (except T, poor drainage) in which *Fusarium* and *Colletotrichum* were used to inoculate the soil, 100 per cent infection was obtained (Fig. 3). From these results, it was reasonably safe to conclude that either *Fusarium* or *Colletotrichum*, or a combination of the two, was the cause of the black scale.

It should be noted that 1 bulb out of 10 in the check (Series M, Table 2) and 1 bulb in each of the *Penicillium*-inoculated soils (Series Q and R, Table 2) were diseased. This perhaps was due to accidental contamination. Although due precautions were exercised in setting up the experiment, all the containers were located on the same bench in the greenhouse, and chance contamination while watering was possible. It is possible also that some of the bulbs used for planting, although appearing perfectly healthy, might have had incipient infections on some of the scales.

The influence of poor drainage on the disease, under the conditions of this experiment, was interesting. In many instances, both in the sterilized and non-sterilized soil lots (series B, D, J, and T), the percentage of diseased bulbs was lower under poor drainage conditions than in the corresponding series where good drainage was provided. This was because soft rot set in under poor drainage conditions. The soft rot either rotted the bulbs completely or, more often, rotted the outer scales (which would normally show black scale lesions) leaving a clean core. It is not claimed, of course, that the artificial poor drainage conditions provided for in this experiment would correspond to poor drainage conditions in the field.

The addition of cottonseed meal to the soil had no effect on the disease under the conditions of this experiment.

While the diseased bulbs (series K, L, W, and X, Table 2) did not recover, they made good growth and produced flowers. This is in agreement with the experience of the growers.

Experiment 2. Soil was taken from Boothville, Louisiana, from a field in which over 80 per cent of the bulbs harvested in 1938 were diseased. The soil was sterilized by autoclaving for 7 hours. The inocula consisted of 12-day-old cultures of the respective fungi (*Fusarium, Colletotrichum,* and *Penicillium*) on autoclaved oats in Mason jars. In this experiment, not only were the 3 fungi used separately and in combination, but also separate series were run with different isolates of *Fusarium* and *Colletotrichum* picked at random. The experiment was started on November 12, 1938, and the bulbs were harvested and examined on August 14, 1939. Ten 6-inch pots were used for each series, and two bulbs were planted in each pot. The pots were kept in the greenhouse during winter, then removed outdoors in April.

The arrangement of the experiment and the results obtained are shown in table 3.

Results. The results of this experiment show rather definitely that of the 3 fungi found associated with black scale, *Colletotrichum* is responsible for the disease. Each of the 4 isolates of *Colletotrichum,* which had been picked at random from many isolations, as well as a combination of the 4 (Table 3, series I–M), produced the disease. Infection in each case was high, the per cent of diseased bulbs ranging from 85 to 100. Of a total of 146 bulbs harvested from the *Colletotrichum* series, 133, or 91.1 per cent, were diseased. This is considered conclusive evidence that the fungus is the cause of the disease.

TABLE 3.—*Pathogenicity studies. Arrangement and results of experiment 2*

Series	Inoculum	No. bulbs harvested	No. bulbs diseased	Per cent diseased
A	Check; non-inoculated	24	0	0.00
B	*Fusarium* (isolate M31A)	30	1[a]	3.33
C	*Fusarium* (isolate M37B)	25	0	0.00
D	*Fusarium* (isolate M49)	23	1[a]	4.35
E	*Fusarium* (isolate M65)	25	0	0.00
F	*Fusarium* (isolate M75B)	35	0	0.00
G	*Fusarium* (isolate M112)	29	0	0.00
H	*Fusarium* (combination of above isolates)	26	0	0.00
	Total of all *Fusarium* series	193	2	0.01
I	*Colletotrichum* (isolate M32)	29	25	86.20
J	*Colletotrichum* (isolate M60)	27	23	85.18
K	*Colletotrichum* (isolate M80)	27	24	88.88
L	*Colletotrichum* (isolate M117)	30	28	93.33
M	*Colletotrichum* (combination of above isolates)	33	33	100.00
	Total for all *Colletotrichum* series	146	133	91.10
N	*Fusarium* + *Colletotrichum* (all above isolates)	21	19[b]	90.48
O	*Penicillium* (3 isolates M38, M69, and M109)	39	4[c]	10.25
P	*F.*, *C.*, and *P.* (all above isolates)	15	11[b]	73.33

[a] Light infection, 1–2 lesions.
[b] Disease less severe than in series I–M where *Colletotrichum* was used alone.
[c] Doubtful, 1–2 lesions per bulb, rot soft, not typical of black scale.

The *Fusarium*, on the other hand, proved non-pathogenic. Six isolates individually or in combination (Table 3, Series B–H), produced no infection. There were 2 lesions on a scale of 1 bulb in series B and 1 lesion on a bulb in series D. These were assumed to be due to accidental contamination by splattering in watering the pots, or to incipient infection on the bulbs planted. Of a total of 193 bulbs harvested from the soil inoculated with *Fusarium*, 191 were healthy.

Four of 49 bulbs grown in the soil inoculated with *Penicillium* (Table 3, Series O) showed a small amount of disease, but the lesions were not typical of black scale; the rot was rather soft and deep.

Experiment 3. This experiment, set up on November 10, 1939, was essentially a repetition of Experiment 2 with the following modifications:

TABLE 4.—*Pathogenicity studies. Arrangement and results of experiment 3*

Series and treatment	No. of bulbs harvested	No. of bulbs diseased	Per cent diseased
A. Check; sterilized soil, no other treatment	1	0	0.0
B. Check; autoclaved oats mixed with soil	5	0	0.0
C–H. Soil inoculated with *Colletotrichum*	8	7	87.5
I–P. Soil inoculated with *Fusarium*	35	5[a]	14.3
Q. Soil inoculated with *Penicillium*	6	0	0.0
R. Soil contaminated with diseased scales	7	5	71.4

[a] Cultures of both *Fusarium* and *Colletotrichum* were obtained from these bulbs.

1. Soil from Baton Rouge, a mixture of equal parts of heavy black clay and sandy loam, was used. This was sterilized by autoclaving; 2. Additional isolates of *Fusarium* sp. were included as inoculum. These were isolated from diseased bulbs from the previous experiments; 3. One series of 10 pots was included in which the soil was artificially contaminated by incorporating in it fragments of diseased scales.

In all, 18 series of 10 pots each were included. The arrangement of the experiment and the results obtained are shown in table 4.

The bulbs used in this experiment came from a "sick" field, and, although selected as being healthy, they had been in contact with infested soil and diseased bulbs; and it was expected that some, at least, would have incipient infections and also be contaminated with spores. Since these bulbs were large, their outer scales were removed and the clean cores were dipped for 5 minutes in a solution of calcium hypochlorite (33 g. per l. of water) a week before planting. This method of disinfection apparently was not entirely effective, because growth of both *Fusarium* and *Colletotrichum* developed on some of the dipped bulbs when they were placed in moist chambers.

Results. The plants were injured severely by freezing. The pots were on a bench near the door of the greenhouse, and on the night of January 18, during freezing weather, the door blew open and the young plants were severely injured. Soft rot set in following the frost injury, and most of the bulbs rotted completely. The results obtained from the surviving bulbs (Table 4) are, however, in essential agreement with those obtained in experiment 2.

Again a high percentage of disease occurred in the soil inoculated with *Colletotrichum*. That some disease occurred in the *Fusarium* series is not surprising, considering the source of the bulbs used. *Colletotrichum* was isolated from the 5 diseased bulbs of the series. The high incidence of disease in the soil contaminated with fragments of diseased scales (Table 4, R) is noteworthy because it illustrates how the fungus is perpetuated in the soil. Lily scales break easily, and many are left in the soil during digging. Furthermore, scales left in the soil usually do not rot but grow and produce new bulblets. For this reason it is very difficult to clean up a field once it becomes infested.

THE FUNGUS

No critical study concerning the identity of the organisms associated with the black scale disease of Easter lily has been found in the literature. The only references found are in the form of notes or brief reports. It is interesting to note that previous to its appearance in Louisiana (5), all reference to this disease either originated in Bermuda or was about specimens of lilies grown in Bermuda. This would indicate that the disease was not known or had not been recognized in other parts of the world. It should be remembered that most of the earlier reports appeared before the virus diseases of lilies (mosaics and yellow flat) had been identified as separate and distinct

entities; and it is evident from the reports that the symptoms and the possible etiologies of the virus and black scale diseases often were confused.

Halsted (1) found *Vermicularia* and *Phyllosticta* on "all the older or browner leaves" of Easter lily plants submitted to him for examination. Stewart (6), investigating what from his description appears to have been fleck mosaic, reported finding *Vermicularia* and mites on the bulbs, "but by no means constantly and seldom in sufficient numbers to account for the damage." Whetzel (8) stated that the black scale is, in some respects, the most serious disease of lilies in Bermuda, and that "the disease is caused by a fungus, but the identity of the pathogene has not yet been determined." In a personal letter (Dec. 8, 1942) Whetzel has kindly informed the writer that the fungus in question, often obtained from the black lesions on the lily scales, was tentatively identified as *Volutella* or *Vermicularia;* and his belief that the fungus was the cause of the disease had been based on the results of inoculation experiments on healthy scales in moist chambers, but that the results of later experiments (unpublished) had satisfied him that the fungus was probably only secondary, the mosaic being primarily responsible for the disease condition and the results described in his published report. It is apparent that Whetzel was not dealing with a single disease; his description of the trouble was obviously a composite picture of the symptoms of two diseases, black scale and fleck mosaic. Ogilvie (2) stated that *Volutella* sp. was the cause of black scale, although in a later report (3) he expressed a different view: "Work on the spotting of the bulbs common in certain soils ('black scale') again showed that this condition is a physiological one due to small points of asphyxiation taking place in heavy or water-logged soils, and cannot be classed as a disease. Such bulbs produce healthy plants. The Volutella sometimes found on the spots is purely secondary."

Waterston (7) named *Colletotrichum gloeosporioides* as the probable cause of black scale, although apparently he had a doubt regarding the identity of the fungus. He stated: "Black scale, ? *Colletotrichum gloeosporioides* Penz. has been provisionally named the fungus responsible for this condition in the bulb and for a premature die-back of the plant in the field."

In a preliminary report (5) the fungus responsible for the disease in Louisiana was tentatively identified as *Vermicularia.* Later, a more critical examination showed that the fungus was definitely not a *Vermicularia* and that it fitted well in the genus *Colletotrichum.* The fruiting body was found to originate beneath the cuticle as a small stroma from which arise the setae which rupture the cuticle. Conidia are produced after the cuticle has been ruptured and the acervulus has been formed (Fig. 4). At no time during its development has the fruiting body a cover of fungus origin.

The fungus grows well in culture. The colonies are at first white, with much aerial growth turning gradually gray and finally nearly black on top with a pinkish tinge in the substratum. In old cultures, the aerial mycelium becomes suppressed and the surface of the colony may become smooth, black,

glistening, somewhat leathery. Young isolates usually produce a great profusion of acervuli on the surface of the agar, with thick, raised masses of conidia through which the setae protrude. Macroscopically, the acervuli at this stage resemble pycnidia. After the isolates have been kept in culture for some time, they usually lose their ability to form acervuli, but they still continue to produce conidia loosely on the mycelium in abundance.

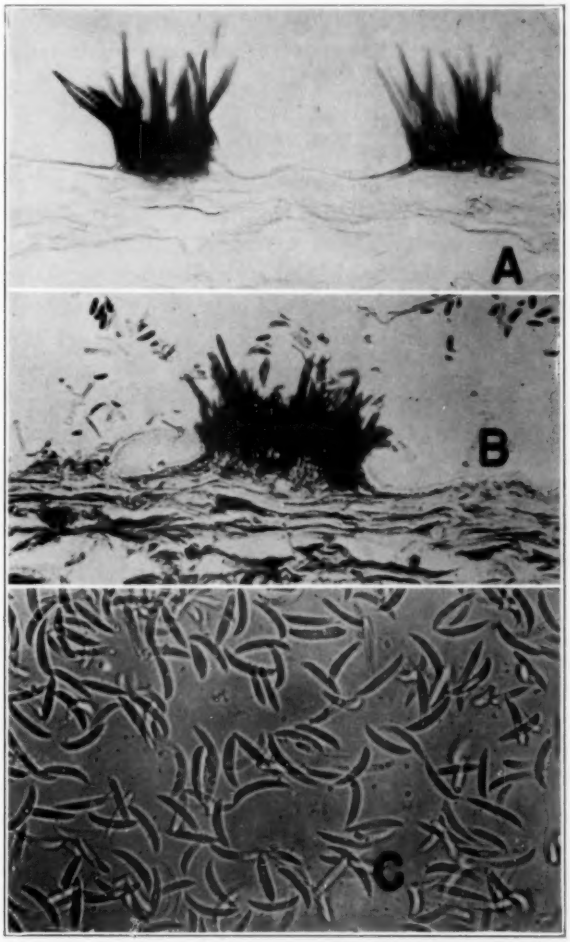

FIG. 4. *Colletotrichum* **lilii**, sp. nov. A. Young acervuli with setae but no conidia. ×200. B. Mature acervulus. ×200. C. Conidia from a 10-day-old culture. ×400.

No description of a *Colletotrichum* on lilies has been found in the literature that fits the causal fungus of the black-scale disease. It is possible, of course, that this fungus has been described on another host, but in view of our present limited knowledge of the pathogenic specificity of many fungi, it is considered advisable to describe it as a new species, for which the following name is proposed.

TECHNICAL DESCRIPTION

Colletotrichum lilii, sp. nov.

Acervuli small, mostly gregarious, arising subcuticularly, on a small stroma, dark brown, densely setose throughout, 43.0–122 μ, ave. 67.2 μ; setae dark brown, rigid, straight, continuous or septate, 29.7–72.6 × 4.0–5.3 μ, average 45.7 × 4.9 μ; conidiophores hyaline, subconical, short, about 10 × 4.5 μ; conidia hyaline, falcate, continuous, subacute, usually vacuolate, 13.2–23.0 × 3.6–4.9 μ, ave. 18.0 × 3.7 μ.

Hab. on scales of bulbs of *Lilium longiflorum*.

Type locality: Plaquemines Parish, Louisiana.

Type specimens: Type specimens, consisting of dried scales, dried agar cultures, and slides, have been deposited in the herbarium of the Botany Department, Louisiana State University (accession No. 4709), and also in the Mycological Collections, Bureau of Plant Industry, Washington, D. C.

ATTEMPTS TO CONTROL THE DISEASE

A few exploratory tests were made over a 2-year period (1938–1939) in an effort to obtain information that might point the way toward effective measures of control. These efforts were directed toward 2 separate objectives, viz: (a) to disinfest the soil in "sick" fields so that healthy bulbs might safely be planted in it, and (b) to disinfect diseased bulbs so that these might be planted in disease-free soil without the danger of spreading the infection to new fields. Unfortunately, all the experiments gave negative results. Some of the treatments were merely ineffective against the disease, but without any harmful effects on the bulbs, while others, in addition to being ineffective, caused varying degrees of injury to the bulbs. The various treatments tried are listed and discussed briefly, even though the results were negative, in the hope that they may serve as a guide in future work on control of this serious disease.

Soil Treatments

The soil treatments were made in 1938 under field conditions in Boothville, Louisiana. These included, (1) chloropicrin at the rate of 2 cc. and 3 cc. per sq. ft. of soil, (2) sulphur ("Toro" brand) at the rates of 700 and 1000 lb. per acre, (3) basic copper sulphate ("Copper Spray 34") at the rate of 1.75 lb. per 25-foot row, and (4) calcium hypochlorite at the rate of 1 lb. per 25-foot row. The sulphur was broadcast and mixed with the soil before the ridges (rows) were made up. The basic copper sulphate and the calcium hypochlorite were applied as narrow bands in the rows and mixed thoroughly with the soil just before planting. All treatments were replicated 3 times in 3 different fields. The soil of fields I and II was of the same type, known locally as "coffee grounds" soil. It was reclaimed land, composed of heavy alluvial soil containing considerable peat-like, imperfectly decomposed vegetable matter. Both fields had a similar history in that both had been planted with lilies the previous year and in both fields practically 100 per cent of the bulbs harvested were diseased. The soil of field III was of the best in that area. It was black, heavy, alluvial with its organic matter well decomposed. This field also was planted with lilies the previous year, and about 50 per cent of the bulbs harvested were diseased, according to the owner. Healthy bulbs were planted in all plots.

Results. All treatments proved ineffective. It is true that in one field all treatments gave significantly higher percentages of marketable bulbs (healthy and slightly diseased) but these differences were not maintained in the other replications. The sulphur and chloropicrin had no effect, harmful or beneficial, on the bulbs. The basic copper sulphate and the calcium hypochlorite, on the other hand, at the rates used, proved somewhat toxic. This toxicity was manifested as delayed emergence, poorer stands, and weaker growth of the plants compared with those of the check and other plots.

The conditions of the experiments were definitely inimical for a fair test of the efficacy of chloropicrin. The soil in the test plots was very heavy, and, in spite of repeated discing and harrowing, large clods were left on the surface that could not be reached by the fumes. No cover was used, and no facilities were available for sprinkling the surface with water to form a seal. Under more favorable conditions, it is possible that chloropicrin might prove effective, for this chemical has been found by many investigators to be a potent soil disinfestant.

TABLE 5.—*Comparison of the corresponding check plots of fields I (diseased scales not removed from the soil) and II (diseased scales removed)*

Field	Healthy bulbs	Lightly diseased bulbs	Severely diseased bulbs	Marketable (healthy plus lightly diseased bulbs)
	Per cent	*Per cent*	*Per cent*	*Per cent*
I	16.3	24.0	59.7	40.2
II	26.4	44.2	29.4	70.6

A noteworthy result of this experiment was the difference in the amount of disease between corresponding plots of like treatments in fields I and II. These two fields, located about one-half mile apart, were as nearly alike as possible. They were of the same type of soil, both were planted to lilies the previous year, and in both approximately 100 per cent of the bulbs harvested in 1938 were diseased. There was, however, this one important difference: In field II, an effort was made to remove from the soil as many as possible of the loose scales, and fragments of scales, that were left from the previous harvest; in field I, no such effort was made, and the soil was thoroughly contaminated with diseased scales. A comparison of the amount of disease in the check plots of fields I and II is shown in table 5. It may be seen that a significantly lower percentage of severely diseased bulbs was obtained from field II than from field I. Differences of about the same magnitude were obtained by comparing other corresponding plots of the two fields. It is believed that less disease occurred in field II simply because most of the infected scales had been removed from the soil, thus lessening the amount of inoculum. If this assumption be correct, then it would seem that cultural practices combining sanitation and rotation would offer a practical way of reducing, if not completely controlling, the disease.

Bulb Treatments

Since infection is relatively shallow, *i.e.*, the fungus does not penetrate deeply into the tissue, theoretically, it might be possible to find some fungicide that would kill the fungus without causing much injury to the bulb. Actually, as brought out by the results of a limited number of trials with various chemicals and combination of chemicals, this does not appear to be so easy. In no case was control obtained, and, in addition, the treatments showed varying degrees of injury to the bulbs. It is admitted, of course, that only a limited number of treatments have been tried, and that neither the list of fungicides, nor the dosages and exposures have been exhausted. It is still possible that an effective treatment may be found, but the results thus far obtained indicate that this line of attack is not particularly promising.

Typically diseased bulbs were secured. The outer, shrivelled scales were removed leaving only the inner live scales, which, however, still contained lesions of various sizes. The bulbs were treated and planted in duplicate sets in sterilized and nonsterilized soil in pots in the greenhouse. Records were kept on the effect of the treatment on germination and subsequent growth. Then, in late summer, the bulbs were dug and examined for presence or absence of disease. These tests were made in 1938 and 1939, as follows:

A. Borax dip, 7.5% solution, 6 minutes at 40° C.

B. Mercuric chloride 1–1000 in 50% alcohol + 1% acetic acid, 3 minutes.

C. Same as B but dipped for 6 minutes.

D. Alcohol (70%) 1 liter; acetic acid 15 cc., mercuric chloride 2 g. Dipped for 10 minutes.

E. Acetic acid 3%, brilliant green 1–20,000, in 50% alcohol, 15 minutes.

F. Bulbs dusted with basic copper sulphate (34% Cu), excess dust screened off.

G. Basic copper sulphate (34% Cu) mixed with the soil at the rate of 150 g. per 50 lb. of soil.

H. Same as in G, but at the rate of 200 g. per 50 lb. of soil.

I. Calcium hypochlorite mixed with the soil at the rate of 200 g. per 50 lb. of soil immediately before planting.

Results. None of the treatments gave any control. The bulbs harvested from all treatments were as severely diseased as those of the checks. In addition, the following toxic effects were noted:

1. Borax. Tip-burn and chlorosis of the lower leaves of the plants after emergence; otherwise growth was normal.

2. The alcohol-acetic acid-mercuric chloride dip for 3 minutes (B) caused no apparent injury.

3. All the rest of the treatments caused injury, manifest in failure of some of the bulbs to emerge and in delayed emergence and weaker, poorer growth of the plants in comparison with those of the checks.

In connection with another disease (mosaic), lily bulbs were treated with hot water, 52° C. for 30 minutes. The bulbs of this lot were not affected with black scale, so no information was obtained on the effect of hot-water treatment on this disease. However, the 30-minute bath at 52° C. was definitely injurious to the bulbs. Some did not grow at all, while others made a decidedly poorer growth than the checks. It is likely, however, that a favorable combination of temperature and period of exposure may be found that will be effective in disinfecting the bulbs without appreciable injury.

SUMMARY

A serious disease of Easter lily, which causes dark-brown to black lesions on the scales of the bulbs in the ground, is described under the name of "Black Scale."

Pathogenicity studies have shown that the cause of the disease is a fungus that fits well in the genus *Colletotrichum*. It is considered to be an undescribed species, and is described as one new to science under the name of *C.* **lilii** sp. nov.

A limited number of experiments consisting of chemical treatments of the diseased bulbs or of infested soil, have been tried. All gave negative results.

DIVISION OF PLANT PATHOLOGY,
LOUISIANA AGRICULTURAL EXPERIMENT STATION,
BATON ROUGE, LOUISIANA.

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STRIPE REACTION OF SPRING BARLEY VARIETIES[1, 2]

H. L. Shands and D. C. Arny

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The stripe disease of barley, caused by *Helminthosporium gramineum* Rabh., has been under observation by workers in the United States and foreign countries for a period of years. When Oderbrucker varieties of barley were grown widely in Wisconsin, stripe often caused yield losses; but with wide-spread usage of Wisconsin Barbless the disease has been less prevalent. Even though stripe has not caused serious yield reductions in recent years, the disease has been given consideration in the general barley-breeding program at the Wisconsin Agricultural Experiment Station. Emphasis has been placed on finding varieties with good agronomic type as well as resistance to stripe and other diseases. Such varieties, used as parental stocks, would likely reduce the breeding problems. New varieties should carry a practical type of resistance, so as to prevent natural stripe epidemics.

EARLIER WORK

Earlier work concerning barley varietal reaction to stripe was carried out by Christensen and Graham (2), DeHaan (3), Fuchs (4), Genau (5), Isenbeck (6), Johnson (7), Majdrakoff (8), Shands (9), Shands *et al.* (11), Stelzner (12), Winklemann (14), and Yu and Hwang (15). The methods of testing varied considerably both in technique and in effectiveness. Shands (9) gave a brief review of the methods of inoculation used. Yu and Hwang (15), used floral inoculation, while the others used the mycelium method for seedling inoculation, except for Stelzner (12) who used a conidial suspension and a partial evacuation method for inoculation of the seed.

In Germany, Isenbeck (6) tested a number of spring and winter barley varieties, using both the floral inoculation and inoculation of germinating kernels. Some of his infections were quite high, but results for individual varieties were not given. Yu and Hwang (15), working in China, have reported detailed readings on the stripe reaction of a large number of varieties. Using conidial inoculation of the flowers between the milky and green-mature stages, their infections averaged 5.4 per cent for the 190 varieties. Their highest 3-year average infection was 51 per cent for Kumflide (C.I. 730). Their local susceptible variety averaged 37.5 per cent for the 3 years. Because a large proportion of the varieties had very low percentages of striped plants, and also because of specialization in *Helminthosporium gramineum*, it is doubtful whether their results could be safely applied under North American conditions. Had flowers been inoculated earlier

[1] These studies were supported in part by funds provided by the Wisconsin Alumni Research Foundation.

[2] Published with the approval of the Director of the Wisconsin Agricultural Experiment Station.

more infection might have resulted. Christensen and Graham (2) tested a smaller number of varieties and obtained rather high percentages of infection, especially with cultural races 33, 34, and 42. Svansota, Manchuria, Velvet, Peatland, and Alpha were relatively susceptible: while Kama-ore (C.I. 694), Trebi (C.I. 936), and the Black Hulless selection were resistant. Stelzner (12), using the conidial-suspension and partial-vacuum techniques of Fuchs (4) and Majdrakoff (8), found some varieties more resistant than others, but his infection percentages were lower than those of several previous workers. Suneson and Santoni (13) florally inoculated a susceptible male-sterile stock that had been previously pollinated with pollen from certain varieties under observation. In this way they compared varieties on the basis of F_1-progeny responses.

Isenbeck (6) tested the reaction of several barley varieties to different collections of the stripe fungus as did Christensen and Graham (2). In these papers it was shown that certain cultures of *Helminthosporium gramineum* are selectively pathogenic and that certain varieties are generally more resistant than others. *H. gramineum* is not so highly specialized as the rusts and smuts, but this specialization should not be overlooked in testing for varietal reaction. However, for practical purposes a few well-selected, highly pathogenic cultures could be used to get a fairly reliable estimate of the reaction of a variety.

METHODS AND MATERIALS

Inoculation

The method of inoculation for varietal tests reported in this paper has been briefly described by Arny and Shands (1). This method is concerned with mycelial inoculation of germinating kernels, and is as follows: After adding sterile water, fine aggregations of mycelium of *Helminthosporium gramineum* are scraped from a 7-day-old slant culture. Two cc. of this suspension are added to each 125-ml. Erlenmeyer flask previously prepared by mixing 15 g. of wheat and 15 cc. of water and autoclaving 45 minutes. When fungus growth on wheat is 5 days old, 100 kernels of the barley variety or selection being tested are surface-treated with 70 per cent alcohol, rinsed in water, and placed in the flask. The contents of the flask are shaken immediately to mix kernels with inoculum and incubated 4 days at room temperature (20–24° C.). Flasks are shaken daily to prevent matting and clumping caused by mycelium and root growth. The number of kernels per test can be varied, although 100 were used for the most part in the varietal tests, and incubation temperature can be lowered if a longer time is used. The entire contents of the flasks are planted in the soil. With this method about 50 per cent of the kernels produce plants in the field, while about 80 per cent produce plants in the greenhouse. In susceptible varieties about 75 per cent stripe infection develops in 6 or 7 weeks time, some infection showing as early as 2 weeks after seeding.

Varieties Tested

A large number of the varieties tested came from the C.I. collection.[3] The seed was originally supplied by R. G. Shands. Previous to the stripe test most of the varieties used had been found resistant in inoculation trials with the 2 sporidium-forming smuts. Also tested for stripe response were selections from the barley-breeding program in progress at the Wisconsin Agricultural Experiment Station. A large number of these hybrid selections do not carry high resistance to the smuts of barley. For the most part selection numbers preceded by "H" were from R. G. Shands.

Cultures Used

The cultures of *Helminthosporium gramineum* were of hyphal-tip origin and date to 1932, 1933, and about 1934 for C–1, 17–1, and 45–2, respectively. These cultures appeared to be stable and pathogenic on a large number of varieties. Culture 17–1 was from a conidium produced on a striped Oderbrucker plant in a row that had been inoculated with culture C–1. Atlas (C.I. 4118) is a variety known to have stripe-infected plants under field conditions in California, and, yet, had no striped plants in 4 artificial inoculation tests reported in this paper. This suggests that some of the varieties, appearing highly resistant under the set of conditions used, may be susceptible in other areas where different conditions of infection might exist. Before assuming a varietal response an investigator should artificially inoculate the varieties in which he is interested, using local cultures and environment. Even though specialization is known to exist, the varietal responses reported herein are thought to be fairly reliable under a wide range of conditions.

RESULTS

Tests have been carried out mostly in the field from 1935 to 1942, although a few were made in the greenhouse. The results of 1936, 1937, and 1939 had lower infection, thereby being less reliable; and because of this only a small part of the data was included. Tests with less than 10 plants were omitted. If more than 50 per cent stripe was found, the variety may not have been tested further; when less than 50 per cent was observed, the variety was tested one or more additional times. In general each variety was tested with at least 2 cultures in any one year.

The results of the several years' tests are combined in tables 1 and 2. Where possible, the C.I. number and the name of each variety are given with the number of tests, the total number of plants observed, low and high per cent infected plants, and the average per cent of infected plants on a weighted basis. The weighted average is determined by use of the total plants in all tests of an individual variety. All of the varieties used in the test are of the spring type, although some are late in heading. Most of the varieties are 6-rowed, rough-awned, with white adhering hulls, and white or

[3] A world collection of barley varieties maintained by the Division of Cereal Crops and Diseases, U. S. Dept. Agriculture.

TABLE 1.—*Stripe reaction of barley varieties when artificially inoculated*

C.I. No.	Name and description[a]	Tests	Total plants	Stripe infected plants		
				Low	High	Weighted average
		No.	*No.*	*Per cent*	*Per cent*	*Per cent*
241	Manchuria	3	117	25	75	42
276	Coast	3	108	13	82	59
531	Hannchen 2	2	136	4	11	7
595	Nepal H, N	2	94	16	16	16
652	Poda	2	64	67	68	68
663	Chile Common	3	114	39	87	65
668	Abyssinian D, P	4	130	22	72	47
918	Korsbyg	7	223	0	9	2
920	White Gatami	8	457	0	2	1
923	Lion (Leiorrhynchum) Bk, S	2	129	9	16	12
934	Odessa	6	270	0	76	23
936	Trebi	9	513	0	25	5
940	Oderbrucker	4	242	58	91	81
967	Jet 2, Bk, N	1	15	.....	.....	93
971	Daniels	2	68	37	96	60
972	Luth	3	147	8	72	46
995	Scarab	4	179	4	30	22
998	Iris N	2	71	89	100	94
1015	Ederle N	2	108	4	7	5
1016	Kwan	3	92	3	35	16
1017	Monte Cristo	3	172	20	39	35
1021	Weider	3	133	0	19	9
1023	Czech	2	137	8	26	16
1024	Quinn	2	137	0	2	1
1027	Black Hulless P, N	6	225	9	37	27
1037	Coventry	2	151	0	0	0
1076	Barquis	2	83	53	59	57
1087	Nani Tal	6	307	0	4	1
1090	Crypt	2	131	25	32	28
1094	Crocket	2	97	19	25	22
1110	Chalet 2	2	138	0	0	0
1111	Chevron	9	458	0	21	7
1118	Featherston	2	149	67	76	71
1137	Oderbrucker	1	22	.....	.....	95
1223	Abyssinian	1	31	.....	.....	84
1243	Abyssinian A	3	53	50	82	70
1248	Excelsior P, N	2	118	87	92	89
1257	Bolivia	2	104	49	66	54
1263	Fleche	2	41	19	28	24
1296	Kitchin D, P	5	203	41	71	62
1312	Lompoc N	2	92	0	0	0
1315	Kusan	2	83	85	97	90
1326	Childs 2, H	2	94	19	43	30
1330	Pannier	2	82	91	93	92
1355	Jusborne S	3	192	35	61	38

[a] All are white or blue, 6-rowed, rough-awned, and have adhering lemma and palea, except where other characteristics are indicated according to the following key:

2 = 2-rowed
I = intermedium
D = deficiens
N = naked kernels
A = Abyssinian intermediate
S = smooth awned
SS = semi-smooth awned
Bk = black
P = purple
H = hooded

Bk and P refer to black or purple color in lemma and palea of threshed grain of hulled barleys and to the color of the caryopsis in naked barley.

TABLE 1.—(*Continued*)

C.I. No.	Name and descriptions[a]	Tests	Total plants	Stripe infected plants		
				Low	High	Weighted average
		No.	*No.*	*Per cent*	*Per cent*	*Per cent*
1370	Spain P, N	2	31	15	28	23
1387	Cape (Transvaal Early)	4	238	1	15	5
1409	McFadden S	1	43	.....	.....	98
1413	Gatami Bk	4	178	0	0	0
1417	Squarehead	1	42	.....	.....	98
1435	Manchuria	1	46	.....	.....	85
1466	Kutan 2	2	58	0	8	4
1468	Burat	2	66	24	50	35
1470	O.A.C. 21	5	243	68	98	83
1473	Manchuria	2	78	44	82	58
1511	Golden Queen	2	59	50	95	80
1517	Oderbrucker	2	68	73	91	79
1529	do. (Wis. Ped. 6)	2	161	53	83	67
1551	Albert	1	47	.....	.....	96
1557	Claude	2	69	65	79	71
1577	Aker	2	62	55	68	60
1603	Ireland A	2	31	20	64	36
1613	Cross	3	108	0	9	2
1614	Fleche	3	143	15	52	36
1615	Ishtar	2	90	0	0	0
1621	Mars	2	96	21	53	37
1622	Osiris	2	105	13	32	23
1692	Mokar	9	469	29	84	69
1698	Reed Triumph	3	206	68	94	76
1969	Chinese Black Bk	4	181	0	0	0
2040	Canada Winter	3	69	0	0	0
2080	White Smyrna 2	3	70	0	17	7
2135	Golden Drop 2	2	128	20	33	26
2201	Eagle	2	52	67	100	75
2203	Boehme's Beardless H	1	30	.....	.....	67
2208	Ethiops Bk, H, N	1	12	.....	.....	92
2213	Nudi haxtoni I, N	2	64	0	0	0
2214	Nudi mortoni I, Bk, N	2	33	12	87	52
2215	Cornutum I, H, N	2	79	0	0	0
2226	Steudelii D, Bk	3	123	10	37	25
2242	Purple Nepal P, H, N	2	36	0	39	20
2244	Surprise	2	66	48	100	64
2253	Kamet Mugi N	2	54	0	0	0
2254	Huwan I	4	129	0	12	3
2259	Manchuria	2	69	67	92	76
2261	Pusa N	2	33	0	0	0
2276	Gatami Bk	4	151	0	2	1
2277	Black Hulless P, N	2	66	29	29	29
2280	Arlington Awnless I	6	206	0	3	1
2282	Pannier	2	33	53	83	69
2284	Black Abyssinian A, Bk	3	122	0	7	2
2286	Nudi haxtoni I, N	2	76	0	0	0
2292	Intermediate I	2	60	45	59	50
2318	Kharsila N	2	41	4	13	7
2319	India N	2	46	0	0	0
2330	Manchuria (Minn. 184)	3	101	66	100	78
2338	Nigrum Bk	2	77	0	3	1
2352	Nudi haxtoni I, N	2	80	0	0	0
2368	Frozoff	1	24	.....	.....	79
2371	.....	2	61	79	100	85

TABLE 1.—(*Continued*)

C.I. No.	Name and description[a]	Tests	Total plants	Stripe infected plants		
				Low	High	Weighted average
		No.	*No.*	*Per cent*	*Per cent*	*Per cent*
2381	..... A	2	74	8	17	12
2382	..... A	2	55	0	17	11
2421	.....	2	73	72	96	81
2432	Rakoff	2	72	8	19	14
2433	Duplex	3	124	25	58	43
2437	Newbly 2	2	99	0	2	1
2448	Himalaya N	2	65	23	43	34
2454	Pegan	2	58	0	7	4
2467	Sampan	4	202	2	13	5
2481	Hanful	2	60	25	58	38
2483	Modia	2	65	0	0	0
2492	Cross	4	235	0	3	1
2494	Manchuria	2	59	73	91	79
2500	Verigat Bk, H	2	37	33	70	43
2505	Englawnless 2	2	30	0	20	7
2509	Englawnless 2	2	51	0	9	2
2511	Plumage 2	2	96	23	36	29
2514	Abyssinian Intermediate A	2	31	16	50	29
2519	.....	2	99	0	0	0
2524	.....	3	141	0	4	3
2542	.....	3	146	2	3	3
2543	.....	2	101	0	2	1
2553	Cap Rouge	2	53	77	90	86
2561	..... S	2	86	38	54	47
2570	..... S	2	67	24	40	31
2572	..... S	2	83	64	64	64
2573	..... S	2	98	0	2	1
2577	..... S	2	92	7	8	8
2589	..... S	2	93	12	14	13
2591	..... S	2	80	15	38	27
2606	..... SS	2	63	0	0	0
2607	 I	2	47	46	52	49
2608	Odessa	2	105	6	33	20
2610	Manchuria	2	104	26	52	40
2618	do.	2	105	60	70	64
2621	Featherston	2	96	75	82	79
2628	Chicago	2	82	71	79	74
2632	.....	2	96	70	95	80
2750	Canadian Lake Shore	2	95	44	57	50
2947	Manchuria (N.D. 2121)	7	377	69	93	86
2982	South Russian	2	125	29	68	47
3124–1	..... D	1	16	.....	.....	63
3187	Regressive 2	2	75	26	41	32
3196	Ehrhardt Frederiksens	2	36	58	75	64
3206–1	.....	2	59	6	26	15
3206–3	.....	2	79	46	63	56
3208–4	.....	4	104	43	93	71
3210–2	.....	6	145	33	73	54
3211–2	.....	1	17	.....	.....	53
3230	..... N	2	62	0	13	7
3245	.....	2	100	30	43	36
3393–2	Bonfarik	2	145	7	7	7
3402	Gujarkhan	4	91	0	6	2
3403	Lyallpur E	3	69	0	24	13
3404	Ludhiana	4	142	13	39	27

TABLE 1.—(*Continued*)

C.I. No.	Name and descriptions	Tests	Total plants	Stripe infected plants		
				Low	High	Weighted average
		No.	*No.*	*Per cent*	*Per cent*	*Per cent*
3408	Delhi	2	70	33	38	35
3410	.....	5	222	0	11	5
3443	India Guzerat	5	225	6	34	19
3485	 2	2	35	0	0	0
3508	.....	2	42	13	21	17
3509	..... N	2	38	26	67	42
3510	..... N	2	46	41	65	50
3530–1	.....	2	74	0	4	1
3552	.....	4	183	0	15	8
3553	.....	4	99	9	44	26
3554	.....	4	240	6	60	19
3555	.....	2	76	2	28	13
3557	.....	2	59	41	85	56
3558	.....	4	191	12	41	19
3559	.....	4	192	0	0	0
3560	.....	2	59	52	78	66
3562	.....	2	61	23	37	31
3570	.....	2	71	2	8	4
3573	 2	3	91	0	7	3
3581	.....	2	80	24	60	38
3582	.....	4	161	12	78	43
3587	.....	2	69	14	44	28
3588	.....	2	72	3	6	4
3614	..... 2, SS	3	106	0	5	1
3617	.....	2	75	21	25	22
3623	 2	3	74	11	50	27
3634	.....	4	151	0	4	1
3635	.....	2	81	0	0	0
3650	.....	2	80	11	15	13
3657	.....	2	80	5	8	7
3659	.....	2	93	0	2	1
3668	 2	3	102	0	14	4
3683	.....	2	72	17	39	25
3694	 2	3	105	0	0	0
3704	 2	3	112	5	46	29
3721	.....	2	66	22	62	43
3724	.....	2	90	0	3	1
3728	.....	4	199	0	15	9
3745	.....	4	167	2	21	8
3746	.....	2	94	11	26	17
3776	.....	4	129	32	72	45
3778	.....	3	118	17	45	25
3780	.....	2	64	0	27	9
3792	.....	2	93	14	86	47
3794	.....	2	81	5	26	15
3797	.....	2	72	2	4	3
3808	.....	4	201	5	17	11
3810	.....	4	182	2	40	14
3816	.....	4	149	7	23	14
3895–1	Revil	4	176	0	0	0
3896–1	.....	4	167	0	11	5
3897–2	.....	4	153	0	3	1
3902–1	Morocco	5	220	0	3	1
3903	.....	5	231	0	50	21
3971–1	.....	2	56	0	12	5

TABLE 1.—(*Continued*)

C.I. No.	Name and description[a]	Tests	Total plants	Stripe infected plants		
				Low	High	Weighted average
		No.	*No.*	*Per cent*	*Per cent*	*Per cent*
4041–2	.....	2	75	3	14	9
4118	Atlas	4	92	0	0	0
4184	.....	2	33	0	0	0
4200–1	.....	2	60	0	0	0
4252	Velvet S	8	419	28	73	45
4289	July	5	111	3	53	43
4425–1	.....	2	98	75	83	80
4428–1	.....	2	108	56	93	76
4500–1	.....	2	94	95	96	96
4502–2	.....	2	105	53	96	72
4559	.....	2	68	3	3	19
4559–1	.....	4	215	26	38	59
4561	.....	4	238	9	50	27
4576–1	.....	2	112	87	94	90
4577	Glabron S	4	235	16	44	30
4577–1	..... S	2	137	15	25	20
4585	Vance 2, S	2	120	0	0	0
4622	.....	4	236	0	2	1
4623	.....	4	191	0	0	0
4637	.....	2	138	36	39	37
4666	Oderbrucker (Wis. Ped. 5–1)	91	4217	61	93	75
4687	Ben Beardless H	2	127	46	47	46
4726	.....	1	17	.....	.....	65
4801	 2	2	129	5	15	10
4821	Dorsett Bk	6	358	0	2	1
4822	..... P, N	2	109	22	44	33
4823	..... Bk	6	337	0	2	1
4827	.....	2	148	35	37	36
4893	.....	2	86	47	85	65
4924	.....	3	142	2	5	3
4951	.....	2	85	25	43	32
5027	Spartan 2, S	3	182	0	0	0
5028	Wis. Ped. 37 S	2	27	13	25	18
5030	Regal	4	196	10	32	14
5072	Mensury	2	57	37	96	60
5105	Wisconsin Barbless (Ped. 38) S	11	737	0	25	14
5220	Harumaki	2	91	5	67	40
5221	Makishu	2	93	0	8	5
5267	Peatland	9	459	24	59	38
5272	Comp. Cross Sel.	2	126	2	9	6
5274	Comp. Cross Sel.	2	76	25	44	32
5346	do.	6	256	10	65	34
5409	do. 2	2	84	18	18	18
5419	do. D	2	42	5	25	15
5459	do. D	2	107	19	21	20
5673	Dryland S	1	39	.....	.....	62
5757	.....	2	91	2	4	3
5788	.....	1	29	.....	.....	69
5817	..... P	1	18	.....	.....	72
5827	..... P	2	46	30	47	37
5899	Murasaki Mochi P, N	2	79	0	0	0
5979	Colsess IV H	9	180	27	86	68
6001	S.D. 1340 S	2	51	29	33	31
6036	Pliter S	2	70	35	50	44
6051	Missouri Early Beardless H	2	57	13	96	68

TABLE 1.—(*Continued*)

C.I. No.	Name and description[a]	Tests	Total plants	Stripe infected plants		
				Low	High	Weighted average
		No.	*No.*	*Per cent*	*Per cent*	*Per cent*
6087	Sanalta 2, SS	2	64	93	94	94
6088	Newal S	7	354	5	30	18
6093	Brandon 1099 S	4	214	17	50	36
6109	Velvon S	3	164	11	24	16
6112	Stewart	2	84	0	0	0
6239	Ioglos S	4	235	17	77	40
6251	Olli	7	199	21	42	37
6352	Cebada 97A	2	90	4	7	5
6492	Manchurian (Wis. 122–3)	4	283	48	79	68
6503	Tystofte Kors	4	169	0	7	3
6531	Persicum 064 (K) 2, Bk, S	3	182	0	0	0
6533	Viner 1163 2	2	131	8	19	14
6544	Abed Juli	2	63	6	36	27
6557	Solenbyg	1	16	.....	.....	63
6572	Brachytic (Minn. 78–4) N	2	93	0	2	1
6614	.....	2	74	86	87	87
6969	Kindred	2	53	79	88	83
6991	Warrior H	4	175	0	8	4
7011	Minn. II–31–19 S	2	78	27	33	30
7015	Minn. II–31–45 S	2	68	11	18	15
7030	Plush S	5	216	23	95	51
7055	Titan 2	2	110	8	13	10
7069	X191–2–1–2 S	6	646	13	37	17
.....	CP170 2	2	143	6	8	7
.....	CP127422 2	4	223	0	6	1
.....	Garton's 3	2	142	76	85	81
.....	HR346	6	350	13	42	31
.....	Michigan 110 2, S	2	94	0	0	0
.....	Trebi sel. (many leaves)	2	101	12	32	21
.....	Olli × Asplund	2	76	13	25	20
.....	S.S. 1446–4	2	54	11	22	17
.....	Tammisto 0432	2	49	25	46	41
.....	WxWx	3	137	65	88	75
.....	Wxwx	3	120	70	92	85
.....	wxwx (waxy)	5	209	79	98	87
.....	Georgine Pedigree 2	2	75	16	42	25
.....	Belgian 2	2	55	13	17	15

blue aleurone. Exceptions to these characteristics are indicated in the column containing the name of the varieties.

DISCUSSION OF RESULTS

An examination of the results in tables 1 and 2 shows that the tests were severe insomuch as a large proportion of varieties have more than 65 per cent stripe. Another measure of the severity of inoculation conditions was shown by the 75 per cent infection of Oderbrucker (C.I. 4666, Wisconsin Pedigree 5–1) which was used as an inoculated check included 91 times in the series of tests. Most of the varieties were given 2 inoculation tests, while some susceptible varieties were given only 1 test, and still others were given as many as 11 tests. The columns carrying the low and high infec-

TABLE 2.—*Stripe reaction of barley hybrid selections when artificially inoculated*

Hybrid selection[a]	Tests	Total plants	Stripe infected plants		
			Low	High	Weighted average
	No.	*No.*	*Per cent*	*Per cent*	*Per cent*
X120–5–26–12–2	7	324	4	67	23
X152–14–2	5	129	0	56	14
X154–2–79	3	168	13	14	14
–14–75	3	114	8	35	25
X156–8–4–4–3–4–1–2	4	193	42	62	55
X173–5–4–1	4	212	39	78	51
–7–1–6	4	170	36	69	51
–10–5–6–1	4	191	52	86	75
X174–10–2–3	3	122	11	58	35
X181–1–2–1	3	119	3	13	6
X181–2–1–2	3	131	2	13	6
X182–2–1	4	132	0	25	17
–8–1–2	3	119	29	39	33
–8–2–8	3	90	40	70	56
–8–3–3	3	152	16	49	46
X182–8–3–4–1	3	71	17	60	45
–8–3–7	4	121	39	74	58
–10–4–4–1	3	76	3	27	12
–16–4	2	84	0	32	20
–21–2–6–1	4	135	10	30	21
X182–28–2	3	128	31	43	39
–43–1	3	74	0	40	18
–79–1–1	4	159	5	25	14
–145–2	5	176	11	33	22
–175–1	3	133	44	69	55

[a] Parentage of hybrid selections are as follows:

X39—Oderbrucker (Wis. Ped. 5) × Lion (Wis. 117)
X60—Lion × Oderbrucker
X102—Oderbrucker (Wis. Ped. 6, C.I. 1529) × X60–1
X105— do. × X39–3–9
X120—X39–6–2–6 × Oderbrucker (Wis. Ped. 6)
X152—X102–2–3–2 × Korsbyg (Wis. 97–3, C.I. 918)
X154—Oderbrucker (Wis. Ped. 5–1, C.I. 4666) × Korsbyg (Wis. 97–3)
X156—X105–2–7–1 × July (Wis. 106, C.I. 4289)
X168—X39–5–8–4–1 × Oderbrucker (Wis. Ped. 6)
X173—X39–9–3–6–8 × Oderbrucker (Wis. Ped. 5–1)
X174—X39–9–3–6–1 × do.
X181—Wis. Barbless (C.I. 5105) × Korsbyg
X182— do. × Olli (C.I. 6251)
X183— do. × C.I. 4561
X184— do. × Peatland (C.I. 5267)
X191— do. × Newal (C.I. 6088)
X202—Newal × Oderbrucker (Wis. Ped. 5–1)
X212—Chevron (C.I. 1111) × X168–5–1
H4—Wis. Barbless × Chevron
H18—X152–14–1 × Chevron
H23—(Wis. Barbless × Chevron) × Wis. Barbless
H25—(Wis. Barbless × Cross, C.I. 2492) × Wis. Barbless
H27—(Velvet, C.I. 4252 × Korsbyg) × Velvet
H35—(X152–14–1 × Chevron) × X152–14–1
H41—Oderbrucker (Wis. Ped. 5–1) × Chevron
H42—Bolivia (C.I. 1257) × Chevron
H47—Chevron × Trebi (C.I. 936)
H80—Chevron × Hillsa (C.I. 1604)

TABLE 2—(*Continued*)

Hybrid selection	Tests	Total plants	Stripe infected plants		
			Low	High	Weighted average
	No.	*No.*	*Per cent*	*Per cent*	*Per cent*
X182–175–2–1	4	116	4	52	20
–202–2	4	136	4	23	12
–243–1	4	141	0	3	1
X183–1	3	103	5	34	29
–5	4	172	5	26	13
X184–4–4	3	138	21	37	34
–5	4	135	6	20	12
–11	2	92	6	9	8
–11–15–1	4	168	6	29	12
X191–2–2–3	5	208	6	37	19
X191–2–3	2	108	5	6	6
–3–2	3	118	2	52	15
–13–1	2	111	15	33	27
–13–2	2	103	20	32	25
–16–2–1	5	170	9	36	26
X191–16–3–1	5	215	12	40	30
–17	3	115	8	67	37
–19–1	3	135	6	37	15
–24	3	132	7	31	18
–25	5	168	20	31	25
X202–1	2	68	15	15	15
X212–1	4	197	3	41	21
H4–3–5–6–2–1–1	4	181	0	18	7
–2	3	146	5	22	10
–3–10–1–1–1	4	129	0	6	2
H4–3–10–4	4	171	10	36	25
–5–1–3–11–4	4	169	4	16	7
–5–1–7–6	5	180	4	15	9
–5–1–12–6–2	2	111	2	11	6
–5–1–12–6–5	3	123	9	16	12
H4–5–6–5–3–2–1	2	99	6	25	18
–5–6–8–3–1–1	4	171	0	13	5
–8–2–2–3–5	2	123	2	6	4
–8–6–1	3	139	6	39	17
–8–7–3	5	270	5	23	14
H4–8–7–3–1–1	4	200	4	36	20
–10–3–6–1–1	4	163	7	24	16
–3–5–6–2–1–3	2	105	5	5	5
H18–3–1–4–1–8	4	151	0	9	4
H23–2–17–4–2	2	147	11	16	13
H25–16–2–1–1	4	176	3	17	12
H27–3–7–4–4	2	124	0	0	0
H35–7–2–1–3	4	138	9	33	20
–7–6–4–1	3	145	27	68	53
H41–4–4–4–1	4	239	8	17	8
H41–4–8–2–1	2	120	8	11	9
H42–5–4–9–9	2	103	61	70	65
–5–4–10–7	2	117	48	53	50
H47–91	2	36	17	22	19
H80–4–2–1	2	72	6	7	7

tions illustrate the variation that was experienced in stripe testing with the method described. Examples: important differences occur between trials with C.I. 241 and C.I. 276, while insignificant differences appear with C.I. 531 and C.I. 652. A lesser number of varieties show identical percentages,

as with C.I. 595. A total of 12 varieties had 90 per cent or more infected plants. They were the following varieties: C.I. 967, C.I. 998, C.I. 1137, C.I. 1315, C.I. 1330, C.I. 1409, C.I. 1417, C.I. 1551, C.I. 2208, C.I. 4500–1, C.I. 4567–1, and C.I. 6087. Stripe symptoms of C.I. 998 were decidedly different from those of the Oderbrucker-Manchuria type. In C.I. 998 striped plants were greatly reduced in size and culm elongation. In contrast striped plants of the Oderbrucker type were only slightly reduced in height. In response to infection, plants of some varieties were distinctly stunted and rosetted, and with little tendency to produce typical long striped lesions on the leaves.

More important from a plant-breeding standpoint, however, are those varieties that resisted the disease. The authors consider that an infection

TABLE 3.—*Source and number of varieties with less than 15 per cent stripe infection or more than 45 per cent*

Source or country	Number of varieties with	
	Less than 15%	More than 45%
Abyssinia	4	8
Afghanistan	1	.....
Asia	2	.....
Asia Minor	1	.....
Australia	2	.....
China	6	1
Chosen	1	.....
Denmark	1	.....
Europe	2	.....
India	8	1
Italy	2	.....
Japan	4	1
Manchuria	6	14
Mesopotamia	1	.....
Russia	3	4
Siberia	1	2
South Africa	1	.....
Sweden	4	1
Switzerland	2	.....
Total	52	32

of 15 per cent or less indicates that a variety is resistant. Thirty-one varieties showed no infection, and 81 more developed less than 15 per cent stripe, making a total of about 112 with resistance. A classification of stripe reaction might be defined as follows: 0–1 per cent = highly resistant; 2–15 per cent = resistant; 16–30 per cent = moderately resistant; 31–45 per cent = intermediate; 46–60 per cent = moderately susceptible; 61–100 per cent = susceptible.

Of the 284 varieties in the C.I. collection tested for stripe reaction and whose eastern hemisphere source is known, 52 varieties had less than 15 per cent stripe and 32 had more than 45 per cent. For convenience, the number and source of these varieties falling into the two classes are given in table 3. Resistant varieties came from different locations of Europe, Asia, and Africa. Manchuria and Abyssinia provided more susceptible types than other countries.

Of the varieties grown commercially in the north-central States Spartan is highly resistant. Hannchen, Regal, Trebi and Wisconsin Barbless are resistant, while Glabron, Newal, Odessa, and Velvon are moderately resistant. Ioglos, Olli, Peatland, Pliter, and Velvet are intermediate, while Dryland, Kindred, the Manchurias, Missouri Early Beardless, and Oderbrucker are susceptible to stripe. Summarized in table 4 are the percentages of stripe obtained in these varieties.

The hybrid selections listed near the end of table 2 vary considerably in their response to stripe, some being resistant, some susceptible, and others

TABLE 4.—*Stripe reaction of some commercial barley varieties of the United States and Canada*

Variety	C.I. number	Average stripe infection
		Per cent
Dryland	5673	62
Glabron	4577	30
Hannchen	531	7
Ioglos	6239	40
Kindred	6969	83
Manchuria, N. D. 2121	2947	86
Manchuria, Minn. 184	2330	78
Manchuria, O.A.C. 21	1470	83
Missouri Early Beardless	6051	68
Newal	6088	18
Oderbrucker, Wis. Ped. 5–1	4666	75
Odessa	934	23
Olli	6251	37
Peatland	5267	38
Pliter	6036	44
Regal	5030	14
Spartan	5027	0
Trebi	936	5
Velvet	4252	45
Velvon	6109	16
Wisconsin Barbless	5105	14

intermediate. Most of the selections are smooth-awned, though a few have rough awns; some have good malting quality; and some have resistance to other important diseases. Most of the smooth-awned selections are deficient in one or more desirable characteristics, and, therefore, would not be considered for replacement of varieties now used for commercial production.

A few of the stripe-resistant varieties that are of interest from the standpoint of their use as breeding stocks are as follows: C.I. 918, C.I. 920, C.I. 936, C.I. 1111, C.I. 1613, C.I. 1969, C.I. 2276, C.I. 2492, C.I. 4821, C.I. 5105, and C.I. 6352. Some of these are particularly valuable because of their resistance to other important diseases, as well as to stripe. Shands (10) has pointed out the value of Chevron (C.I. 1111) as a breeding stock, since it carries resistance to stem rust, mildew, scab, and stripe.

SUMMARY

Over a period of 8 years 375 barley varieties and selections were tested for their reaction to the stripe disease by bringing germinating barley kernels into contact with the mycelium of *Helminthosporium gramineum* Rabh.

Some varieties were highly resistant to the cultures of the fungus used in these tests; other varieties were intermediate in their stripe reaction; while still others were susceptible. Oderbrucker (C.I. 4666), which was used as the inoculated check 91 times in the series of tests, averaged 75 per cent stripe-infected plants. This indicates that the method of inoculation was effective. Physiologic specialization of the fungus may influence varietal response to inoculation. The host response to stripe infection may differ distinctly between varieties.

Some of the stripe-resistant varieties may be of value as breeding stocks because they have other desirable characters.

AGRICULTURAL EXPERIMENT STATION,
MADISON, WISCONSIN.

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INHERITANCE OF CHLAMYDOSPORE AND SORUS CHARACTERS IN SPECIES AND RACE HYBRIDS OF TILLETIA CARIES AND T. FOETIDA[1]

C. S. HOLTON[2]

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The demonstration of hybridization between *Tilletia caries* (DC.) Tul. (*T. tritici* (Bjerk.) Wint.) and *T. foetida* (Wallr.) Liro (*T. levis* Kuehn) by Flor (2) suggested the possibility of studying the inheritance of certain physiological and morphological properties of these organisms, such as pathogenicity, chlamydospore markings, and spore ball characters. That the expression of these characters is genetically controlled is shown in the results of experiments reported by several investigators, although the manner of their inheritance has not been determined. For example, Flor (2) observed that species-hybrid spores were smooth, like the *T. foetida* parent, and Hanna (4) made similar observations. However, neither of these workers observed segregation into smooth and reticulate spores in the progeny of the hybrid spores. Furthermore, Hanna (4) states that the spore balls of the hybrids resembled the *T. foetida* parent spore balls and that the characteristic trimethylamine odor of the *T. foetida* parent was present in the hybrids. Apparently, however, no segregation for these characteristics was observed in any of the hybrids. Likewise, Becker (1) found culture characters to be of a heritable nature but the manner in which they are inherited was not determined, although the inheritance of pathogenicity was found to be intermediate in one cross and recessive in another.

In 1938 (7) the writer described a hybrid-race of *Tilletia caries*, derived from a cross between T–9 (*T. caries*) and L–8 (*T. foetida*). The F_1 spores of this hybrid were reticulate, although the reticulations were less prominent than those of the *T. caries* parent. In this hybrid, therefore, the reticulate character was at least partially dominant. No segregation of spore characters was observed. Later, however, studies (8) with hybrids produced by crossing the dwarf bunt with L–8 and T–12 revealed that the hybrid spores were intermediate between the parent types in the F_1 and that segregation occurred in the F_2. This was the first published evidence of segregation of factors for chlamydospore characteristics in hybrids of the bunt fungi. More recently (10), a report on the inheritance of pathogenicity was published in which it was shown that factors for pathogenicity and chlamydospore characters are inherited independently. The purpose of

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[2] Pathologist, Division of Cereal Crops and Diseases, Bureau of Plant Industry, Soils, and Agricultural Engineering, Agricultural Research Administration, U. S. Department of Agriculture.

this report is to present some detailed observations on the inheritance of factors governing chlamydospore and spore-ball characters in certain hybrids between *T. caries* and *T. foetida*.

MATERIAL AND METHODS

Fifty-five hybrids were used in these studies, of which 32 were hybrids between *Tilletia caries* and *T. foetida* and 23 were hybrids between races within these species. The races used were, T–8, T–9, T–10, T–12, L–7, L–8 (9), and dwarf bunt (8). Hybrids were produced by the method already described (6), each one being designated by number, and pedigreed as to race, chlamydospore, and sporidium.

Chlamydospores of the F_1 generation were obtained from Hindi (C.I. 8454) wheat, which had been inoculated with paired monosporidial lines. Attempts to culture complete sets of individual sporidia from F_1 spores

TABLE 1.—*Summary of observations on chlamydospore characters of hybrids between Tilletia caries and T. foetida and their respective races*

Races crossed	Number of hybrids	Number of hybrids with F_1 spores		Remarks[a]
		Smooth	Reticulate	
L– 8 × T– 9	6	3	3	Reticulations small.
L– 7 × T–10	8	4	4	Reticulations medium to large.
L– 8 × T– 8	1	1	0	Spores uniformly spherical (like T–8 parent).
L– 8 × T–12	7	0	7	Reticulations small.
T– 8 × T– 9	9	0	9	Reticulations large and variable in shape.
L– 8 × L –7	2	2	0	Spores irregular in shape.
T– 8 × T–10	4	0	4	Reticulations medium to large.
T–12 × Dwarf	8	0	8	Reticulations medium to large.
L– 8 × Dwarf	10	0	10	Reticulations medium to large.
Total	55	10	45	

[a] The reticulations on the chlamydospores of all of the *T. caries* parents were large.

were unsuccessful and for this reason chlamydospores were used for inoculum to produce the F_2 generation on the susceptible winter wheat variety Hybrid 128 (C.I. 4512). Obviously, chlamydospore material obtained thusly was not readily adapted to an exact factorial analysis. Nevertheless, it was suitable for a study to determine the nature and extent of variation in chlamydospore characters of hybrids between species and races. Microscopic observations were made, therefore, to determine the dominant or recessive nature of certain chlamydospore characters in the F_1 and the nature and extent of segregation of these characters in the F_2.

Spore balls of certain races of the bunt fungi differ primarily in size and shape. The expression of these characters, however, usually is governed to some extent by the variety infected. Furthermore, considerable variation within a race may be expected, owing to the difference in the location of the bunt balls on the spike. Consequently, in studying spore-ball characters of parent races and hybrids between them, the spore balls were

selected from the same variety and the same relative position on the spikes, usually the central portion.

EXPERIMENTAL RESULTS

Chlamydospore Characters

The observations on chlamydospore characters in the F_1 generation are summarized in table 1. Twenty-four of the interspecies hybrids had reticu-

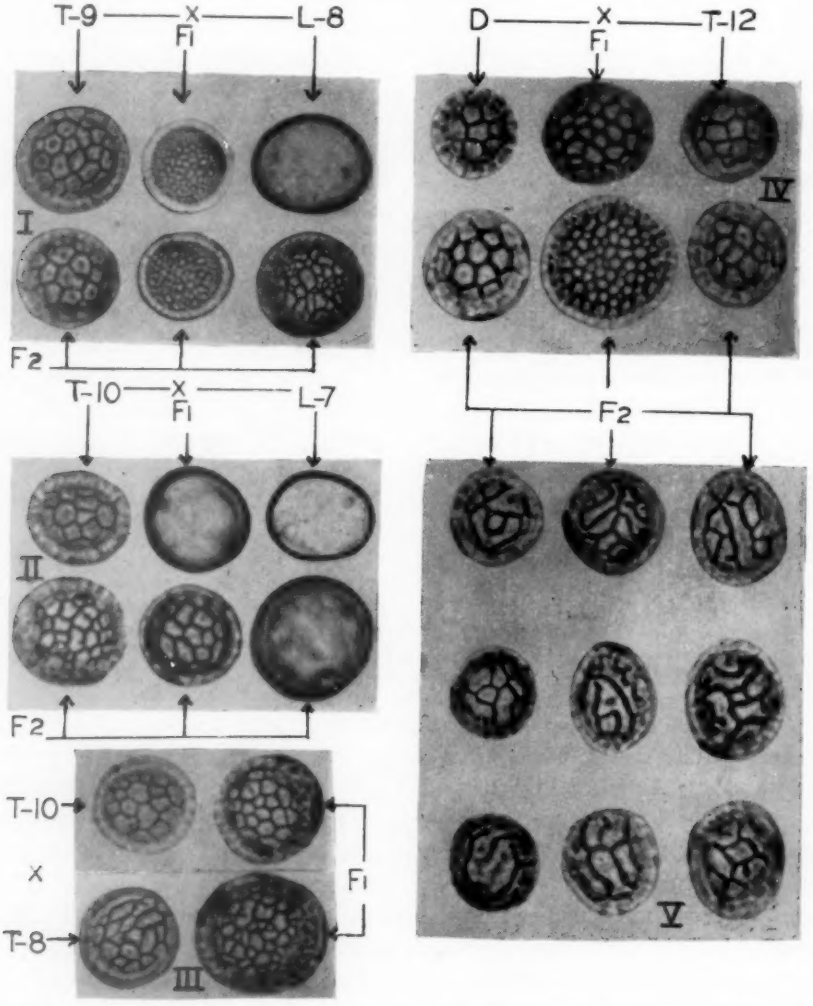

FIG. 1. Photomicrographs of parent and hybrid chlamydospores of *Tilletia caries* and *T. foetida* showing heritable nature of spore markings. (Enlarged and retouched). I. Races T–9 × L–8. II. Races T–10 × L–7. III. Races T–10 × T–8. IV. Dwarf bunt × T–12. V. Cerebriform F_2 segregate from F_1 of IV. × about 850.

late spores and 8 had smooth spores. The spores of hybrids between races of *T. foetida* were smooth and those of hybrids between races of *T. caries*

were reticulate. As indicated in table 1 and illustrated in figure 1, the reticulations of the hybrid spores were highly variable in character. For example, in hybrids between L–8 and T–9 the reticulations were extremely small (Fig. 1, I) and the reticulations of the spores of hybrids between L–8 and T–12 were similar in character. On the other hand the reticulations of the spores of several other species hybrids ranged in size from medium in some to large in others. This suggests lack of complete dominance of the reticulate character in any of the interspecies hybrids studied. Furthermore, in hybrids between races of *T. caries*, there was lack of complete dominance of reticulation type represented by different races. This is indicated by the wide variation in size and shape of reticulations on F_1 chlamydospores of hybrids between T–8 and T–10 (Fig. 1, III) and the reticulations of F_1 chlamydospores of hybrids between the dwarf bunt and T–12, which were distinctly different from those of either parent (Fig. 1, IV). As already stated, 8 of the interspecies hybrids had smooth F_1 spores, indicating complete dominance in these hybrids of the smooth character of the *T. foetida* parent (Fig. 1, II).

Although there was considerable variability in chlamydospore markings in the F_1 generation of some of the hybrids, the nature and degree of variability in the F_2 was clearly indicative of segregation of factors that control these characters. For example, in certain hybrids between T–9 and L–8 some of the F_2 spores resembled the T–9 parent, some resembled the F_1 spores, and others had reticulations that were highly variable in character (Fig. 1, I). The L–8 parent type (smooth spores) was not recovered in the F_2. On the other hand, in hybrids between T–10 and L–7 which had smooth F_1 spores, both parent types in addition to other types were recovered in the F_2 (Fig. 1, II). Similar results were obtained with hybrids between the dwarf bunt (D) and T–12 (Fig. 1, IV). Worthy of special note is the cerebriform type illustrated in figure 1, V. Although not present in all sori, this type of spore was predominant in some of them. They have not been found to be viable.

Sorus Characters

That sorus characters are genetically controlled, at least to some extent, is indicated by the results of a study with Hybrid 39, which resulted from a cross between T–9 and L–8. The spore ball types of the parents and hybrids are illustrated in figure 2. It will be noted that there is only a slight difference in the sori of T–9 and L–8 on the variety Hybrid 128 while the sori produced by Hybrid 39 (F_6) on this variety are much smaller than those of these two races. Furthermore, the hybrid sori are smaller on Hohenheimer than the T–9 parent sori and smaller on Oro than the L–8 parent sori. Thus it appears evident that certain factors governing the size of the sori were operative in this hybrid between T–9 and L–8, which resulted in the production of sori smaller than those of either parent. Furthermore, the fact that the hybrid sori were smaller than the parent

sori suggests transgressive inheritance. Also there appears to be a slight difference in the shape of the sori of the hybrid and L–8 on Oro (Fig. 2). This is regarded as some evidence that sorus shape likewise is genetically controlled. Similar observations have been made in connection with other hybrids.

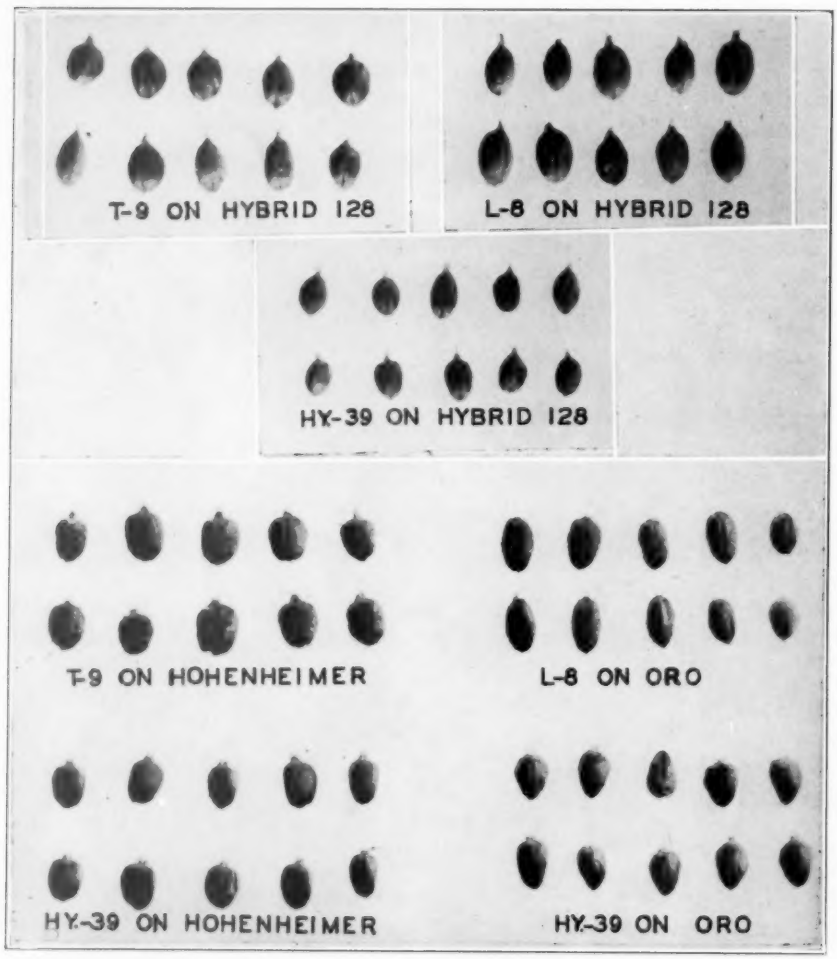

FIG. 2. Type of sorus produced on 3 varieties of wheat by races T–9 and L–8 and a hybrid between these races.

DISCUSSION

The exact nature of inheritance of factors governing chlamydospore and sorus characters could not be determined in this study, mainly because complete culture sets of primary sporidia from individual chlamydospores could not be obtained. Nevertheless, there is conclusive evidence in the data and observations reported that chlamydospore markings and size of

the sorus are determined by genetic factors and that wide variations in these characters probably are the result of different combinations of genetic factors. Consequently, one might expect to encounter variable types in collections of bunt from commercial wheat fields, inasmuch as hybridization might occur under natural conditions. This is especially true with regard to the reticulations of the chlamydospores of *Tilletia caries*, which, in the hybrids studied, were found to range in size from extremely small to relatively large.

That hybrid spores have been observed in field collections is indicated by the reports of several investigators in different parts of the world. In the United States, Flor (2) found *Tilletia caries* and *T. foetida* occurring together in field collections in the majority of cases, suggesting ample opportunity for hybridization. Furthermore, he observed all degrees of reticulation on spores in field collections, some spores being so finely reticulated as to be difficult to distinguish from the smooth spores of *T. foetida* while others were so coarsely reticulated as to appear spiny. Gassner (3) observed spores that had much finer reticulations than *T. caries* and thin spore walls like *T. foetida*. He regarded this as an intermediate type that resulted from hybridization, and designated it as *T. caries intermedia*. It occurred infrequently (only 8 heads in over 2000 were found) and usually in the presence of the 2 main species, in several cases all 3 being in the same head. This association seems to support Gassner's theory that the intermediate type is the result of interspecific hybridization. Intergrading types, such as those described by Flor, apparently were not observed by Gassner (3). Hirschhorn (5), however, describes and illustrates several types of reticulations, intergrading from the typical *T. caries* type to Gassner's *intermedia* type. She suggested that these intermediate types resulted from hybridization between *T. caries* and *T. foetida*. This suggestion is logical, since the experimental evidence now at hand indicates that the atypical spores observed by Flor (2), Gassner (3), and Hirschhorn (5) did result from hybridization between the two species. Furthermore, from the taxonomic viewpoint, the importance of recognizing this possibility is emphasized by the fact that Savulescu *et al.* (11) established the species *T. triticoides* as distinct from *T. caries* on the basis of its slightly smaller spore size and more delicate reticulations. In other words, because of the intergrading types of chlamydospores that result from hybridization, delimitation of species of *Tilletia* on wheat according to size of reticulations can scarcely be justified.

SUMMARY

In some hybrids between *Tilletia caries* and *T. foetida* the smooth character of the *T. foetida* spores was completely dominant over the reticulate character of the *T. caries* spores. In other hybrids the reticulate character was partly dominant over the smooth character. Variability in type of reticulations and size of spores was observed in the F_1 generation of certain hybrids between races of *T. caries*.

Segregation and recombination of factors for chlamydospore characters occurred in the F_2 generation of hybrids between *Tilletia caries* and *T. foetida* and between races within species. Spores resembling both parents and spores distinctly different from both parents usually were recovered in the F_2. In one species hybrid smooth-type spores of the *T. levis* parent were not recovered in the F_2.

Evidence is presented indicating that size and shape of the sori of *Tilletia caries* and *T. foetida* are, at least partly, genetically controlled.

AGRICULTURAL EXPERIMENT STATION,
PULLMAN, WASHINGTON.

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PRELIMINARY REPORT ON SOME MOSAIC DISEASES OF IRIDACEOUS PLANTS

FLOYD F. SMITH[1] AND PHILIP BRIERLEY[2]

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Comparatively few members of the Iridaceae are reported subject to mosaic diseases, and none of these plants are recorded as suscepts of the well-known viruses of wide host range. Iris mosaic (2) has been shown to be transmissible by sap, and by the aphids *Macrosiphum solanifolii* (Ashm.) and *Myzus persicae* (Sulz.), but the known host range is limited to the genus *Iris*. A mosaic disease of gladiolus, described by Dosdall in 1928 (4), has assumed greater importance in recent years (3). Although shown to persist in vegetative parts, gladiolus mosaic has not previously been shown to be transmissible. A virus disease of Freesia, not known to occur in the United States, has been described briefly and attributed to Freesia virus 1 by Smith (6), but no report of transmissibility has yet appeared. Atanasoff (1) and later observers have reported mosaic symptoms in Crocus.

Corms of Tigridia (*T. pavonia* (L.f.) Ker) were received from a grower in western Washington in 1939, with the report that a mosaic-like disease had spread rapidly through a previously normal planting. Mosaic symptoms are common in gladiolus varieties grown at the Plant Industry Station, Beltsville, Maryland, and elsewhere. Similar mottling symptoms have been noted in plants received from commercial sources, including *Babiana* hybrids, *Ixia* hybrids, *Sparaxis* hybrids, *Streptanthera cuprea* Sweet, *Tritonia lineata* (Salisb.) Ker, *Tritonia* hybrids, including *Montbretias*, and *Watsonia marginata* (L.f.) Ker. Stocks from New Jersey, Ohio, and California dealers appear similarly affected, indicating that the virus or viruses are generally distributed in the United States. A study of the interrelationship of these virus diseases, and of their possible relationship to such diseases of other monocotyledons, was begun in 1940. Although the work is far from complete, transmissibility of mosaic viruses in *Tigridia*, *Gladiolus*, and several other genera may be reported at this time.

SYMPTOMS

Symptoms of the diseases in the several source plants had the following characteristics:

Tigridia mosaic, appearing in the greenhouse in plants grown from corms received from the Washington grower, consisted of pale-green to yellowish-green, irregular streaks and blotches (Fig. 1, A) in leaves and flower bracts. Breaking of the flower color in the form of pale, white streaks or, less com-

[1] Entomologist, Division of Truck Crop and Garden Insect Investigations, Bureau of Entomology and Plant Quarantine, Agricultural Research Administration, United States Department of Agriculture.

[2] Pathologist, Division of Fruit and Vegetable Crops and Diseases, Bureau of Plant Industry, Soils, and Agricultural Engineering, Agricultural Research Administration, United States Department of Agriculture, Beltsville, Md.

monly, dark streaks, has been detected in *Tigridia*, but these symptoms have been less striking than the breaks commonly present in the flowers of mosaic-affected bulbous iris.

A number of gladiolus plants of the variety Picardy were selected for flower breaks in the field in 1941 and grown under glass in 1942. Young leaves developing in April and May showed a fine-grained, angular, green mottling, and the flowers showed distinct breaking in color. In this sample

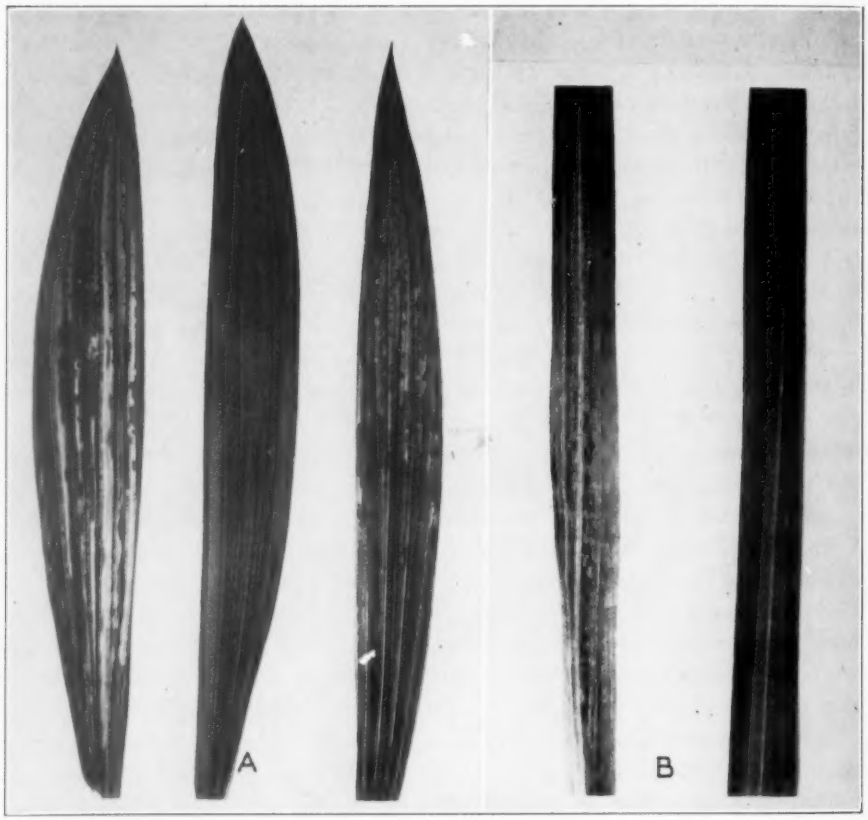

FIG. 1. A. Tigridia mosaic. Left to right, natural infection, control, and experimentally produced infection (*Aphis gossypii* transfer). B. Gladiolus mosaic. Experimentally produced (*Myzus persicae* transfer) and control.

of Picardy the flowers were pink in bud, but became pale-streaked or spotted when full-blown or while fading. Both color-intensified and bleach patterns were noted in other lots of this variety and in other varieties. The corm symptoms noted by Dosdall (4) have not been detected in our material.

Characteristic mosaic mottling appeared in plants of the other genera mentioned above. A bright-yellow, angular mottle was present in naturally infected *Ixia* hybrids, light and dark green mottling in the others. Darker "tear-drop" flower breaks were noted in *Babiana* hybrids, and pink streak

breaks were present in white flowers of affected *Ixias*. *Sparaxis* hybrids exhibited a strong green leaf mottling, with frequent crinkling and pinching of affected leaves not observed thus far in other iridaceous plants.

TRANSMISSIBILITY

The carborundum leaf-rubbing technique failed to transmit Tigridia-mosaic virus to Tigridia seedlings, or gladiolus-mosaic virus to gladiolus seedlings. The methods of culturing and transferring aphids were those described in connection with other studies (5). Control seedlings of *Tigridia, Gladiolus,* and *Tritonia crocata* (L.) Ker have remained healthy in the screened and fumigated greenhouse throughout this study.

The data in table 1 show that Tigridia-mosaic virus is transmissible by *Aphis gossypii* Glover. Transfers by *Macrosiphum lilii* (Monell) and by *Myzus circumflexus* (Buckt.), indicated by single infections by each species, also are considered authentic, as no evidence of contamination was detected in the greenhouse-grown seedlings under test. Mosaic mottling typical of the source material appeared in experimentally infected plants, but no flower breaks were observed.

Gladiolus-mosaic virus was transmitted to seedling gladiolus plants by *Myzus persicae* in three trials (Fig. 1, B) and by *M. circumflexus* in one trial. Symptoms appeared in 2 to 4 weeks in actively growing plants, or else in the second season. Flower breaks like those in the Picardy source appeared in the 6 experimentally infected plants that bloomed. Uninoculated seedlings developed no breaks like the virus type, but some of these produced flowers with fine striping or marbling believed to be heritable color patterns.

Tritonia crocata seedlings offered some promise as test plants for iridaceous viruses. This species is readily grown from seed under glass, but is not ideal in symptom expression. Some exploratory transfers to this species are listed in table 2. *Myzus persicae* transmitted virus from mosaic-affected *Babiana, Ixia, Sparaxis, Streptanthera, Tigridia, Tritonia,* and *Watsonia* to *Tritonia crocata* seedlings. Mottling appeared at the base of young leaves 2 to 8 weeks after exposure, as fine, angular, dark-green islands on a paler green ground. No flower breaks were detected in *Tritonia.*

No certain distinctions between viruses transferred from different genera can be made from these data (Tables 1 and 2). Apparent differences noted in vector relations, ease of transfer, or incubation periods may well be due to differences in growth rate of inoculated seedlings or to other factors. In other tests, however, some differences in symptom expression indicate that distinct viruses or virus strains are concerned in iris mosaic from German iris and in the mosaic of *Sparaxis. Myzus persicae* transferred from mosaic-affected German iris to gladiolus seedlings produced a prominent rectangular mottle pattern in 2 weeks, the 2 affected shoots, of 5 exposed, later turning yellow, then brown, and dying prematurely. When this test was repeated with the same vector, small side shoots were mottled and killed in

a similar sequence in 4 pots of 5 exposed, but larger plants in the same pots remained unaffected. Apparently a virus from mosaic-affected German iris is lethal to gladiolus, whereas the gladiolus-mosaic virus from Picardy

TABLE 1.—*Transmission of virus from mosaic-affected Tigridia to Tigridia seedlings, and from mosaic-affected gladiolus to gladiolus seedlings, by aphids*

Date of transfer	Aphid species	Aphids per plant	Seedling plants infected[a]	Minimum incubation period[b]
	Tigridia mosaic			
		Number		*Days*
May 29, 1941	*Aphis gossypii*	25	3/9	20
June 19, 1941	do.	25	2/5	21
June 1, 1942	do.	20	3/6	26
June 21, 1941	*Aphis rumicis* L.	20	0/10	.....
May 28, 1942	do.	25	0/5	.....
May 29, 1941	*Macrosiphum lilii*	20	1/8	340
June 23, 1941	*Myzus circumflexus*	20	1/3	17
	Gladiolus mosaic			
July 30, 1942	*Macrosiphum solanifolii*	30	0/10	.....
Aug. 26, 1942	do.	10	0/4	.....
May 28, 1942	*Myzus circumflexus*	25	2/5	252
May 28, 1942	*Myzus persicae*	25	4/5	25
June 26, 1942	do.	25	4/6	221
Feb. 5, 1943	do.	40	5/5	14

[a] Number infected over number exposed.
[b] Days until symptoms appeared.

TABLE 2.—*Transmission of virus from mosaic plants of several iridaceous genera to Tritonia crocata seedlings by Myzus persicae*

Date	Source of virus	Aphids per test	Plants infected[a]	Minimum incubation period[b]
		Number		*Days*
Dec. 31, 1942	Babiana hybrid	80	7/16	14
Feb. 5, 1943	Gladiolus (var. Picardy)	240	0/20	.....
Feb. 5, 1943	*Iris germanica* L.	240	0/20	.....
Dec. 31, 1942	Ixia hybrid	80	2/20	14
Jan. 30, 1942	Sparaxis hybrid	256	1/10	56
Jan. 2, 1943	do.	80	2/20	48
Feb. 5, 1943	do.	240	0/20	.....
Dec. 31, 1942	*Streptanthera cuprea*	80	11/15	14
May 28, 1942	*Tigridia pavonia*	150	1/5	19
Feb. 5, 1943	do.	240	0/20	.....
Dec. 31, 1942	Tritonia hybrid	80	4/18	14
Dec. 31, 1942	*Watsonia marginata*	80	3/20	50

[a] Number of plants infected over number exposed.
[b] Days until symptoms appeared.

induces mild mottling and flower breaks only when transferred by the same vector.

Transfers of *Myzus persicae* from mottled *Sparaxis* produced positive results but no unusual patterns in *Tritonia crocata*. A transfer to gladiolus

seedlings yielded mosaic mottling in 3 of 5 exposed, 1 of which developed a prominent, rectangular, yellow pattern similar to the yellow mottling occasionally seen in gladiolus in the field. Transfers by *M. persicae* from *Sparaxis* to Clara Butt tulip repeatedly induced white spotting in leaves and cucumber-mosaic-type flower breaks. Mechanical transfers to tobacco from such inoculated tulips, and also from the *Sparaxis* source, yielded a cucumber-mosaic virus strain similar to those commonly present in Easter lilies affected with necrotic fleck. No cucumber-mosaic virus was recovered by mechanical methods from the gladiolus showing exceptional symptoms following inoculation from *Sparaxis*.

In further mechanical transfers cucumber-mosaic virus isolated from *Sparaxis* produced white-ring and watermark primary lesions in Turkish tobacco, followed by systemic mild yellow mottling. In White Spine cucumber, primary lesions appeared in the cotyledons, as was the case with the celery strain, followed by mild yellow mottling but no stem necrosis. *Zinnia elegans* Jacq. developed systemic mottling. White-spot primary lesions of the cucumber-mosaic-virus type were produced in *Mesembryanthemum crystallinum* L.

DISCUSSION

The occurrence of cucumber-mosaic virus in *Sparaxis* is believed to be a new record for the family Iridaceae, as well as for this genus. No evidence has been obtained thus far that other genera of Iridaceae are susceptible. The role of this virus in the mosaic disease, observed to occur naturally in *Sparaxis*, cannot be evaluated until healthy individuals are available for inoculation. The evidence at hand suggests that this virus may contribute to the effects observed but that it is not the only virus concerned in the mosaic disease of *Sparaxis*.

It is uncertain whether gladiolus mosaic, Tigridia mosaic, and the similar diseases observed in several other iridaceous genera are caused by a common virus. The differential response of gladiolus seedlings indicates that iris-mosaic virus, as found in German iris, is distinct. Until these relations are more definitely established, it is considered unwise to propose technical names for the viruses concerned. Common names such as gladiolus-mosaic virus and Tigridia-mosaic virus should be adequate at present.

SUMMARY

Tigridia-mosaic virus was found to be transmissible by *Aphis gossypii*, *Macrosiphum lilii*, and *Myzus circumflexus*, but not by sap; gladiolus-mosaic virus was transmissible by *M. circumflexus* and *M. persicae*, but not by sap.

Virus was transmitted from mottled *Babiana*, *Ixia*, *Sparaxis*, *Streptanthera*, *Tigridia*, and *Watsonia* to *Tritonia crocata* by *Myzus persicae*.

Cucumber-mosaic virus was found naturally occurring in *Sparaxis* hybrids.

PLANT INDUSTRY STATION,
BELTSVILLE, MD.

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RHIZOCTONIA LEAF SPOT OF COTTON

D. C. Neal

(Accepted for publication January 20, 1944)

A leaf spot of cotton, apparently due to the Rhizoctonia fungus, was reported from Louisiana in July, 1943 (3). The disease was first observed by H. B. Brown, Agronomist of the Experiment Station, on leaves of an unknown strain of Deltapine cotton plants growing in a bottom field near a drainage ditch. Subsequently, specimens of the affected leaves were collected and referred to the writer for examination for a possible disease-producing organism. Microscopic study and pure-culture isolations from affected leaves have shown that the Rhizoctonia fungus is responsible for this leaf spot. Also, cultural characteristics and growth relationships indicate that it is in all probability the common soil species, *Rhizoctonia solani* Kühn (*Corticium solani* (Prill. & Del.) Bourd. & Galz.). The disease also was later observed on other varieties, including Coker and Delfos cotton. While frequently a considerable area of the leaf is attacked, causing some shedding, observations made so far show that damage to the plant is not serious enough to cause much reduction in yield.

DESCRIPTION OF THE DISEASE[1]

The disease is readily recognized. In the early stage, light brown, irregularly shaped spots of varying size appear between the veins, bordered by a dark-purplish ring—a distinctive feature. As the fungus advances, it causes the tissues immediately surrounding the spots to become chlorotic, and the necrotic area of the old spot cracks or falls out, presenting a ragged, shot-hole appearance (Fig. 1). Microscopic and macroscopic examinations reveal abundant light- to yellowish-brown mycelium over and around the spots on the lower surface of the leaves. The mycelium agrees closely with morphological characteristics described by Duggar (2) for *Rhizoctonia solani.* Hyphal strands are frequently numerous and may easily be observed with a hand lens.

CULTURAL STUDIES AND GROWTH ON MEDIA

The fungus was easily isolated by sterilizing for brief intervals (2 to 3 minutes) small sections of infected leaves in a 3.5 per cent B–K stock solution (active ingredient calcium hypochlorite) diluted with 2 parts water and subsequently plating on potato-dextrose agar. By using newly affected leaves, especially the chlorotic tissue surrounding the spots, the fungus may be obtained in practically 100 per cent of the platings by this technique. In addition to potato-dextrose agar, the fungus also grows well on Trommer's malt agar, the hyphal strands becoming cinnamon-brown or darker after 6 or 7 days' growth in Petri plates and forming pseudo-sclerotial wefts.

[1] The writer is indebted to Dr. E. C. Tims of the Department of Plant Pathology, Louisiana Agricultural Experiment Station, for the photographs of this leaf spot.

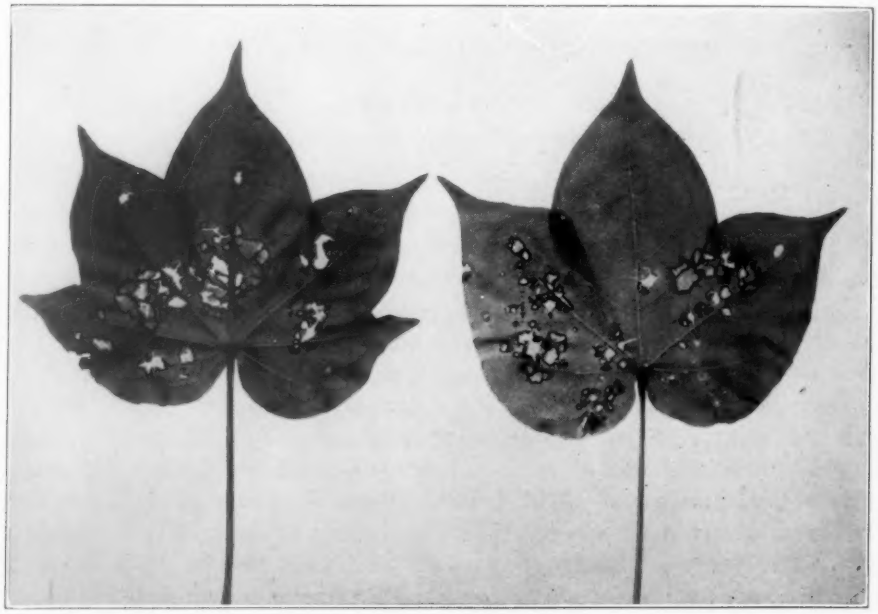

FIG. 1. Cotton leaves infected with *Rhizoctonia* fungus. Note shot-hole appearance of leaves and dark-purplish ring surrounding spots. Natural infection. Variety—Deltapine. Photographed July 16.

PATHOGENICITY TESTS

Typical lesions of the disease have been produced on leaves of plants by inoculating with agar cultures, and the fungus later reisolated from the inoculated leaves. In most cases 3- or 4-day-old cultures of the fungus on potato-dextrose agar in Petri plates were employed for the inoculations, a block of the agar culture approximately a half inch square being applied with scalpel to the upper and lower surfaces of separate leaves. As controls, other leaves were treated similarly with sterile agar blocks. The results of these inoculations are shown in table 1 and illustrated in figure 2.

TABLE 1.—*Number and per cent of cotton leaves with lesions 10 days following inoculation with Rhizoctonia isolate*[a]

Variety	Leaves inoculated			Leaves with lesions			Per cent[b] infection
	Surface placement of inoculations			Surface placement of inoculations			
	Upper	Lower	Total	Upper	Lower	Total	
Coker	10	10	20	6	10	16	80.0
Deltapine	10	10	20	4	8	12	60.0
Delfos	6	6	12	4	6	10	83.3
Coker control	....	....	0	....	....	0	0.0
Deltapine control	....	....	0	....	....	0	0.0
Delfos control	....	....	0	....	....	0	0.0

[a] Inoculated July 24.

[b] Average per cent infection all varieties upper surface inoculations 53.8, lower 92.3.

Inconspicuous lesions were noticed on some of the inoculated leaves after 8 and 9 days, respectively; therefore, under favorable weather conditions, such as light rainfall and moderate temperatures, which prevailed during these experiments, the period of incubation is perhaps 6 or 7 days.

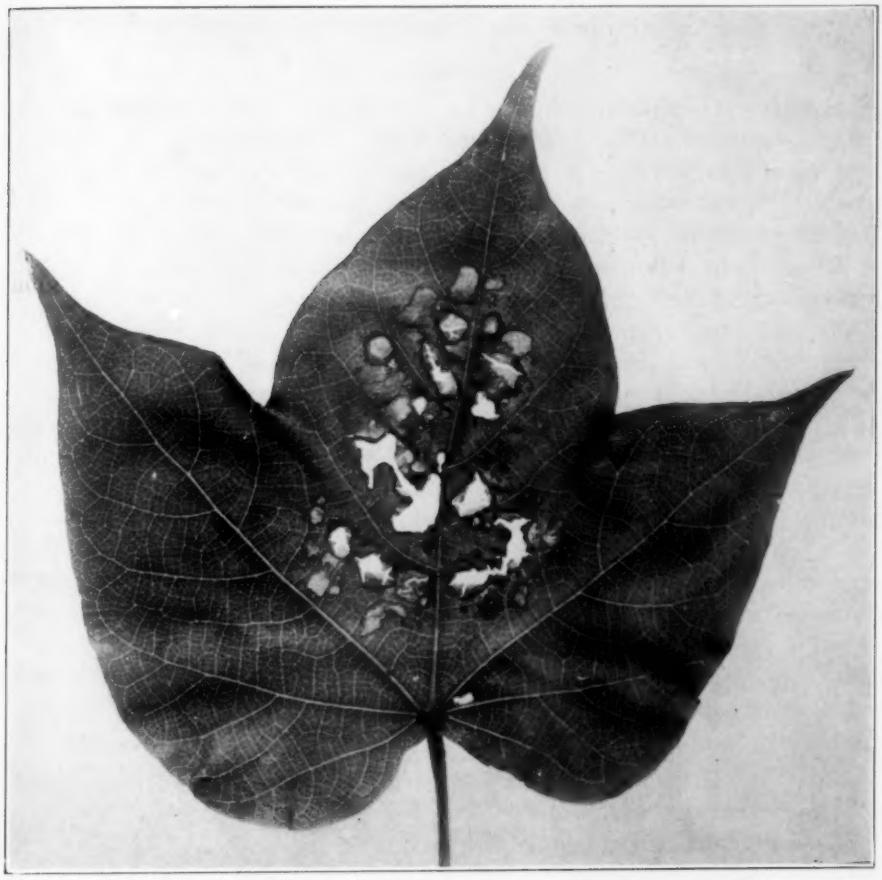

FIG. 2. Cotton leaves artificially infected with *Rhizoctonia* by placing sections of agar cultures of the fungus on the uninjured leaf surface. Inoculated July 26. Photographed August 3. Variety—Deltapine.

DISCUSSION

Although *Rhizoctonia solani* is known to cause "damping off" and "sore shin" of cotton seedlings (1) and sometimes persists when cold, wet weather continues through late spring, causing injury to root systems and stems of older plants (4), this is the first report, as far as the writer is aware, of leaves of mature cotton plants being attacked by a species of *Rhizoctonia*.

In studying possible sources of infection and means of dissemination of this fungus to the leaves of large cotton plants, a search was made of all other vegetation growing in close proximity, such as grasses and weeds

None of these hosts, however, gave evidence of being a carrier of the disease. Also, none of the cotton plants showed the Corticium stage of the fungus, thus eliminating probable spread by basidiospores. It would seem, therefore, that infection of the leaves may have occurred through dissemination of infested soil particles or debris, by wind, or other agencies. These points are still under investigation.

SUMMARY

A leaf spot of cotton, hitherto unreported and caused by the Rhizoctonia fungus, occurred at Baton Rouge, Louisiana, during the summer of 1943. It developed in mid-July on Deltapine plants, and later was found in other near-by fields on other varieties, including Coker and Delfos.

Leaf symptoms are distinctive and are described herein.

The fungus was readily isolated from infected leaves and its pathogenicity established through inoculation experiments. Under conditions favorable for infection, such as cloudy weather and moderate temperatures, the period of incubation was estimated to be approximately 6 or 7 days.

Cultural studies of this leaf-spot *Rhizoctonia* indicate that its morphology and growth characteristics are similar to and probably identical with the common soil fungus, *Rhizoctonia solani* (*Corticium solani*). Although certain possibilities present themselves, the exact manner by which it is disseminated to the leaves of mature cotton plants is unknown.

While the disease was frequently found attacking large leaf areas, sufficient to cause shedding, it has not been found yet to be of serious economic importance.

U. S. DEPARTMENT OF AGRICULTURE,
AGRICULTURAL RESEARCH ADMINISTRATION,
BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING,
DIVISION OF COTTON AND OTHER FIBER CROPS AND DISEASES,
IN COOPERATION WITH THE
LOUISIANA AGRICULTURAL EXPERIMENT STATION,
BATON ROUGE, LOUISIANA.

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SOME WAYS BY WHICH NUTRITION MAY AFFECT SEVERITY OF DISEASE IN PLANTS[1]

G. M. Shear and S. A. Wingard

(Accepted for publication January 27, 1944)

The purpose of this paper is to call attention to some experimental evidence obtained by other workers, and by the writers, that brings out possible relationships between certain unbalanced conditions in the mineral nutrition of a host plant and the severity of attack by certain vascular pathogens. That the nutrition of plants affects their susceptibility to certain diseases has been shown by numerous investigators and their findings have been reviewed by Wingard (8). In many instances the effect of nutrition seems to result from its effect on the general vigor of the plant. In other cases deficiencies of certain elements affect the susceptibility of a plant to a disease, while deficiencies of other essential nutrients have no apparent effect, thus showing that other factors are involved in such cases. These effects are not always easily explainable, although their pathological, as well as physiological, implications are of unquestioned importance.

The findings of Spencer and McNew (7) regarding the effects of nutrition on the susceptibility of sweet corn to *Phytomonas stewarti* show very clearly certain ways in which nutrition affects the development of a disease. They found that seedlings dwarfed by high concentrations of nitrogen, phosphorus, or potassium were more severely infected than those grown at concentrations more conducive to rapid growth, and that seedlings deficient in either nitrogen or phosphorus were only slightly infected whereas potassium-deficient seedlings were severely infected. Regarding the effect of potassium, they state that the disease was more severe in seedlings deficient in potassium than in those supplied with low concentrations of potassium and that this difference in severity may possibly be explained as follows: The pathogen may have a low potassium requirement and may cause considerable injury when inoculated into seedlings weakened by a deficiency of potassium. However, with the addition of small amounts of potassium, the seedlings start normal growth, and in so doing may become more resistant.

As a result of further study on the effect of nitrogen on the corn wilt organism, McNew and Spencer (4) showed that the amount of nitrogen in the tracheal sap of maize directly affects the growth of *Phytomonas stewarti*. This direct effect they state is probably attributable to the fact that the bacterium lives almost entirely in the tracheal tubes during the early stages of its invasion and, therefore, depends upon the materials in the transpiration stream for its sustenance. They present conclusive evidence, also, that the corn wilt bacterium depends on inorganic nitrogen in the tracheal sap for its parasitic existence.

[1] Published with the approval of the Director, Virginia Agricultural Experiment Station, as Scientific Paper No. 120.

The recent findings of Shear (6) regarding the effects of different amounts of fertilizer on the nutrient content of the conductive tissue of tobacco explain more fully the results obtained by Spencer and McNew and also throw some light on other ways by which nutrition may affect the susceptibility of a plant to disease. Increasing the amount of a nutrient in the fertilizer generally increased the amount of that nutrient in the conductive tissue. A deficiency of an element in the soil showed a corresponding decrease in the amount of that element in the conductive tissue of the plant.

However, the deficiency of a particular element sometimes resulted in a marked increase of the relative amounts of other elements in the conductive tissue, and this may be of importance in explaining the effect of a nutrient deficiency on the severity of infection by certain vascular parasites. When nitrogen, for example, was left out of the fertilizer, nitrate nitrogen in the conductive tissue was found to be very low, while phosphorus was found to be high and potassium not significantly affected. These findings agree with those of Carolus (1), who worked on certain truck crops and indicate that when nitrogen is deficient, protein formation is interfered with and the phosphorus that would otherwise be used in making protein compounds is accumulated in the conductive tissue. Apparently, however, the degree of nitrogen starvation was not great enough to interfere appreciably with carbohydrate metabolism in which the postassium was utilized.

In the tobacco experiments, when phosphorus was left out of the fertilizer, phosphorus in the conductive tissue was found to be very low, but the amount of nitrates and potassium was not affected thereby. These findings differ from those of Carolus (1), and Eckerson (2), working with tomatoes, who found that there was an accumulation of nitrate nitrogen in phosphorus-deficient plants. Eckerson explains that there is a decrease in the reductase activity in phosphorus-deficient tomato plants, which inhibits the utilization of nitrates. MacGillivray (3), on the other hand, found that there was an increase in the percentage of nitrogen in phosphorus-deficient tomato plants but that it was confined to water-soluble forms other than nitrates. These apparently conflicting results may be explainable on the basis of the degree of phosphorus deficiency obtained in the different studies. The phosphorus requirements of the various plants studied may have varied also.

When potassium was left out of the fertilizer mixture used in the tobacco experiments, potassium in the conductive tissue was found to be low, while phosphorus and nitrate nitrogen were high. Carolus (1) mentions the increase in nitrate nitrogen when potassium is deficient, but does not mention any effect that it has on the amount of phosphorus. Nightingale, Schermerhorn, and Robbins (5) found that a lack of potassium in tomato, beet, and lettuce plants retards the synthesis of organic nitrogen from nitrates. This might explain the high amounts of both nitrate and phosphorus found in the conductive tissue of tobacco. If the nitrates were not converted under

conditions of potassium deficiency, the utilization of phosphorus would be impeded the same as it is when nitrogen is deficient.

The preceding findings provide a basis for a logical explanation of the increased severity of wilt infection of corn seedlings deficient in potassium. It would appear, therefore, that the increased wilt severity is due to an increased rate of bacterial multiplication caused by the increase in nitrate nitrogen in the conductive tissue, resulting from the potassium deficiency. When potassium deficiency is less severe, more of the nitrate in the corn plant would be utilized by the plant itself, thus reducing the amount in the conductive tissue available for the growth of the wilt organism. This in turn would lessen the severity of the effects caused by the parasite.

Such a direct relationship between the nutrition of the host and the parasite as that suggested above would not occur in many instances, as most parasitic organisms require more complex nitrogen compounds for their nutrition. Assuming our interpretation to be correct, if the severity of infection caused by a disease-producing organism were found increased by addition of an excess of nitrogenous fertilizer, but not increased by a deficiency of potassium, the inference would be that the pathogen probably could not directly utilize nitrate-nitrogen within the host. It might perhaps indicate that the severity of infection was increased either by the succulence of the host tissue or more probably by the presence of an abundance of some more complex nitrogen compound, essential to the growth of the parasite, which was being produced in excess of the requirements of the host.

SECTION OF BOTANY AND PLANT PATHOLOGY,
VIRGINIA AGRICULTURAL EXPERIMENT STATION,
BLACKSBURG, VIRGINIA.

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COMPARATIVE STUDIES OF TWO CARROT LEAF DISEASES[1]

W. J. HOOKER[2]

(Accepted for publication January 29, 1944)

INTRODUCTION

In Wisconsin, as well as in many other areas, carrots (*Daucus carotae* L.) are attacked by 2 leaf-spotting fungi, *Cercospora carotae* (Pass.) Solheim and *Macrosporium carotae* Ellis and Langlois. It has been noticed in Wisconsin and has been reported in Massachusetts (1) that the seasonal cycles of these diseases differ from each other. *C. carotae* occurs most commonly during July and early August in Wisconsin, whereas *M. carotae* develops in severity during August and throughout September. This study was initiated to determine whether the difference in seasonal cycle could be explained on the basis of the response of the 2 fungi to temperature or to other factors.

METHODS

Single-spore cultures of *Macrosporium carotae* did not sporulate on potato-dextrose agar, and as a result a medium containing 6 per cent fresh carrot leaves and 2 per cent agar was used for spore production. It was later shown that 2 per cent water agar induced sporulation equally well. Both media supported only meager vegetative growth. *Cercospora carotae* was grown on a medium prepared with carrots, cucumber, and agar described by Mrak, Phaff, and Douglas (2).

Seedling carrots of the Danvers Half Long Variety in the 4- to 6-leaf stage were used in greenhouse studies with the exception of 1 experiment in which leaves developed from storage roots of the Chantenay variety were used. Leaves were inoculated by spraying with a spore suspension. The plants were held in moist chambers for 48 hours, and then they were returned to the greenhouse bench. The houses in which inoculated plants were incubated were held at constant temperatures of 16, 20, 24 and 28° C., respectively. Each inoculated leaf was placed in 1 of 4 classes designated as healthy and slightly, moderately, or severely diseased, respectively. From such data a disease index was calculated in which the 4 classes were given equal weight. Thus the index for any given set of plants might vary from 0, indicating all were healthy, to 100, in which all were severely diseased. In certain cases the data were treated by the analysis of variance method. The writer is indebted to Dr. J. H. Torrie for advice in connection with this phase of the study.

SYMPTOMS OF THE TWO DISEASES

Differences in symptoms of the 2 diseases have been described by Doran

[1] The writer is indebted to Dr. J. C. Walker for advice during the course of this investigation and in the preparation of the manuscript.

[2] Formerly Instructor in Plant Pathology, University of Wisconsin; at present Research Associate in Botany, Iowa State College, Ames, Iowa.

and Guba (1) and those of *Cercospora carotae*, described and illustrated by Thomas (3). The distinction between the diseases is best shown in figure 1.

FIG. 1. A, *Macrosporium carotae;* and B, *Cercospora carotae* on leaves of carrot. *M. carotae* generally produces dark, irregularly shaped lesions whereas those produced by *C. carotae* have a light-tan-colored center and tend to be more nearly circular in shape.

The spots caused by *C. carotae* are, in general, more nearly circular than those caused by *Macrosporium carotae*. Leaf tissue is invaded more rapidly

by the latter fungus resulting in more irregularly shaped lesions which may quickly involve the leaflet tip or the entire leaflet. The leaf spot caused by *C. carotae* is generally a light tan color, especially if the disease has developed during periods of relatively low humidity, as was the case with the leaf shown in figure 1; whereas, if development has occurred in a moist atmosphere, the entire lesion may be dark colored and not so nearly circular, as illustrated by Thomas (3). Lesions produced by *M. carotae* are generally dark-brown to black, and chlorosis of surrounding tissue is considerably more prominent than in the case of spots caused by *C. carotae*. Both fungi attack the petioles, producing elongate lesions that often result in death of the entire leaf. Petiole lesions in the case of *C. carotae* often have a more or less light-colored center, whereas those caused by *M. carotae* are dark in color.

TEMPERATURE RELATIONS IN PURE CULTURE

Throughout the investigation spore germination and growth responses in Petri-dish cultures were determined on potato-dextrose agar. Spore sus-

TABLE 1.—*Effect of temperature on germination of spores of Macrosporium carotae and Cercospora carotae*

Temp.	*Macrosporium carotae*				*Cercospora carotae*			
	Experiment number			Average	Experiment number			Average
	1	2	3		1	2	3	
°C.	*Per cent*	*Per cent*	*Per cent*	*Per cent*	*Per cent*	*Per cent*	*Per cent*	*Per cent*
4	0	0	0	0	0	0	0	0
8	0	0	0	0	8	8	12	9
12	36	12	72	40	50	91	26	56
16	91	10	88	63	92	91	84	89
20	92	85	100	92	100	96	98	98
24	100	79	95	91	100	97	100	99
28	100	93	100	98	98	99	100	99
32	100	100	100	100	98	93	100	97
37	.....	96	86	91	0	0	0	0

pensions were placed in Petri dishes at room temperature in experiments 1 and 2, whereas in experiment 3 the spore suspension was added to plates previously brought to incubator temperature. Observations were made at the end of about 3 hours with *Macrosporium carotae* and at the end of 24 hours with *Cercospora carotae*. The percentage of germination at different temperatures is shown in table 1. In the case of *M. carotae* germination was practically complete over a range of 20 to 37° C. during the 3-hour period of incubation. Slight reduction was noted at 16°, marked reduction at 12°, and no germination at 8 and 4°. Observations after 1- and 2-day incubation showed good germination at both 4 and 8°, while at 37° good germination occurred with no further mycelial development. Practically complete germination of *C. carotae* occurred at 20 to 32°; it was slightly reduced at 16°, and markedly so at 8°. No germination was observed at 4°, but, after a week, short germ tubes had developed, while during this interval none had

developed at 37°. These results are in general accord with those of Doran and Guba (1) and Thomas (3).

For studies of growth, cultures in dishes were inoculated in the center with a heavy suspension of spores in the case of *Cercospora carotae* and with a 5-mm. disk of mycelium and agar in the case of *Macrosporium carotae*. Measurements of colony diameter were made at the end of 1 week. Relative mycelial growth of both fungi in culture is shown in figure 2. Both fungi showed a distinct growth optimum at 28° C.

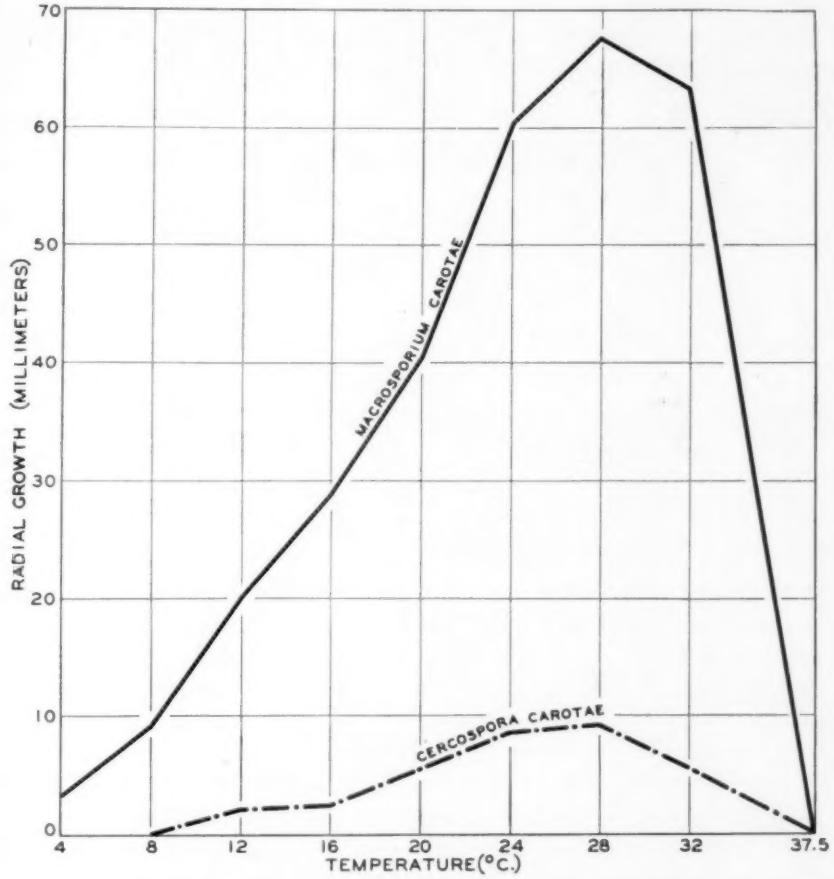

FIG. 2. Temperature relations of *Macrosporium carotae* and *Cercospora carotae* as indicated by colony diameter after incubation for 1 week on potato-dextrose agar at temperatures indicated. Graph drawn by Eugene Herrling.

INFLUENCE OF TEMPERATURE ON DISEASE DEVELOPMENT

In order to distinguish between the effect of temperature on infection and on subsequent disease development, 2 distinct series of inoculations were carried out. In the first case, the temperature of inoculation was maintained the same as that of the house in which disease development was to take place by putting individual moist chambers over the plants in the respective tem-

perature houses. In the second case, plants were inoculated in a common moist chamber and then removed to their respective temperature houses. The optimum temperature for seedling development of Danver's Half Long carrot was about 24° C.

The results of inoculation experiments in which inoculation and subsequent disease development occurred at the same temperature are shown in table 2. Markedly slower disease development by *Macrosporium carotae* occurred at the low temperatures. In all trials the disease developed more slowly at 16° C. than at 20°. Although this difference was not significant at the 5 per cent level, it was marked. Significant differences were obtained when disease development at 16 or at 20° was compared with that at 24 or

TABLE 2.—*Disease development by Macrosporium carotae and Cercospora carotae on leaves of carrot at temperatures indicated*

Organism	Temp., °C.	Disease index				
		Replication number				Average
		1	2	3	4	
Macrosporium carotae	16	28	11	6	10	13.75
	20	53	25	13	15	26.50
	24	51	44	49	59	50.75
	28	55	25	47	52	44.75
Cercospora carotae	16	15	12	12	2	10.25
	20	24	32	37	12	26.25
	24	41	43	52	53	47.25
	28	42	53	67	53	53.75
Difference required for significance (19: 1)						15.6

at 28°. A slight but not significant reduction in disease occurred in 3 of the 4 trials between 24 and 28°. Although chlorosis did not develop consistently, both diseases showed greater chlorosis at 16 and 20° than at 24 and 28°. In general, infection by *M. carotae* resulted in more chlorosis than was the case with *Cercospora carotae*.

The trends with *Cercospora carotae* were, in general, more clearly defined than are those shown by *Macrosporium carotae*. Disease development increased progressively with temperature. Significant differences at the 5 per cent level were obtained between 16 and 20° C. and between 20 and 24°. The difference between 24 and 28° was not significant.

When another series was run in which all the plants were inoculated at 20° C., and the plants then incubated at 16, 20, 24, and 28°, the same relation of temperature to disease development maintained. It may be concluded, therefore, that there is little or no difference to be found between the 2 fungi in their response to temperature as measured by spore germination, growth in pure culture, or in the progress of pathogenesis at constant temperature levels. The difference in seasonal cycles of the 2 diseases must, therefore, be based on other factors.

RELATION OF AGE OF LEAF TO DISEASE DEVELOPMENT

It was observed during the temperature studies that *Cercospora carotae* developed most severely on the younger leaves, whereas *Macrosporium carotae* showed a preference for the older leaves. Doran and Guba (1) inoculated excised carrot leaves with *M. carotae*. After 3 weeks they observed an average of 24 infection points on old leaves for each point of infection on young leaves. They reported that young leaves were susceptible to *C. carotae*, but apparently they did not observe that young leaves were more susceptible than old leaves. In this investigation, plants in the 4-leaf stage were used. Leaves were numbered from 1 for the youngest to 4 for the oldest. The results given in table 3 show the striking difference between

TABLE 3.—*Influence of leaf age upon development of carrot leaf blight and leaf spot*

Organism	Leaf No.[a]	Disease index						
		Replication number						Average
		1	2	3	4	5	6	
Macrosporium carotae	1	6	0	0	12	19	25	10.3
	2	37	12	31	12	50	31	28.8
	3	56	37	37	50	37	75	48.7
	4	69	50	69	62	87	94	71.8
	Difference required for significance (19: 1)[b]							28.0
Cercospora carotae	1	75	62	75	37	58	75	63.7
	2	75	19	37	31	50	58	45.0
	3	37	19	31	25	17	42	28.5
	4	19	19	37	25	8	37	24.3
	Difference required for significance (19: 1)							26.7

[a] Leaves numbered from the youngest to oldest on seedling carrots.
[b] The variances within treatments were homogeneous by Bartlett's test for homogeneity.

the 2 fungi. *M. carotae* increased very decidedly in severity from the youngest to the oldest leaf while with *C. carotae* the reverse was true.

SUMMARY AND CONCLUSIONS

The leaf blights of carrot have been studied under controlled conditions to determine what might be the underlying causes of the striking contrast in season cycles of the 2 diseases. Spores of both fungi germinated over a wide range of temperature with optimum for both at 28° C. Low temperature (16° C.) retarded disease development with both, while there was an increase in disease index with increase in temperature with both up to 24° C. It is thus apparent that temperature has little to do with determining the early-seasonal severity of *Cercospora carotae* and the late seasonal development of *Macrosporium carotae*.

When age of leaf was studied in relation to disease index, a wide contrast was to be found. *C. carotae* was most severe on young leaves, and patho-

genicity decreased markedly on older leaves at time of inoculation. *M. carotae*, on the contrary, was least pathogenic on young leaves and most severe on old leaves. This difference in the relative resistance of the host leaves to the 2 fungi may account in large measure for the fact that one disease is at its peak in mid-season when high temperature and young foliage favor it, while the second finds a more congenial substrate later in the season as more leaves approach maturity even though temperatures at that time are slightly less favorable.

DEPARTMENT OF PLANT PATHOLOGY,
UNIVERSITY OF WISCONSIN,
MADISON, WISCONSIN.

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PHYTOPATHOLOGICAL NOTE

The Nomenclature of the Broomcorn Millet Smut Fungus.—In the interests of stability of nomenclature the Federal Department of Agriculture follows the International Rules of Botanical nomenclature in its publications. This will bring about changes in a number of fungus names heretofore in common use and this is particularly true in the case of names for the cereal smut fungi. However, only in one instance has it been found necessary to set up a new binary name and that involves the broomcorn millet smut fungus, now known in American practice as *Sphacelotheca panici-miliacei* (Pers.) Bub.

Persoon in his *Synopsis Fungorum* (1801), which is the starting point for the nomenclature of the smut fungi, lists the millet smut fungus as a subspecies, *panici-miliacei*, of his *Uredo segetum*, and this is the name which has been more commonly used as a specific epithet. However, Schlechtendahl in 1824 validly published *Caeoma destruens* as a name for this species, citing Persoon's name, and thus *destruens* becomes the first legitimate specific epithet available under the provisions of section 11, article 58, of the rules. It does not appear that transfer to the genus *Sphacelotheca* has been made previously and in order that the name may be available, the necessary new combination is here proposed, with pertinent synonymy appended.

Sphacelotheca destruens (Schlecht.) comb. nov.

Uredo segetum subsp. *panici-miliacei* Pers. Syn. Fung., p. 224. 1801.

Uredo carbo var. *panici-miliacei* DC. Flore Franç. 6: 76. 1815.

Caeoma destruens Schlecht. Fl. Berol. 2: 130. 1824. Link in Willdenow Sp. Plant. 6 (2): 3. 1824.

Uredo destruens Duby Bot. Gall. 2: 901. 1830.

Tilletia destruens Lev. Ann. Sci. Nat. III. 8: 372. 1848.

Ustilago panici-miliacei Wint. in Rab. Krypt. Fl. 1: 89. 1884.

Sorosporium panici-miliacei Takahashi Bot. Mag. Tokyo 16: 184,247. 1902.

Sphacelotheca panici-miliacei Bub. Houby Česke 2: 27. 1912.

—John A. Stevenson and A. G. Johnson, Bureau of Plant Industry Station, Beltsville, Maryland.

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